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Skin Diving
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William O. Lodge

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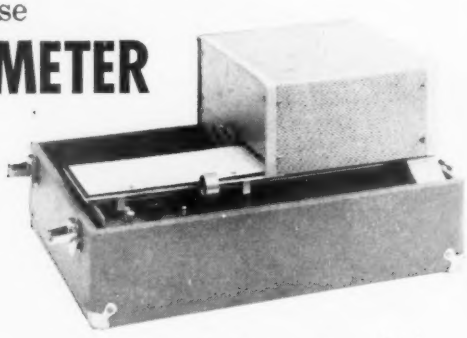

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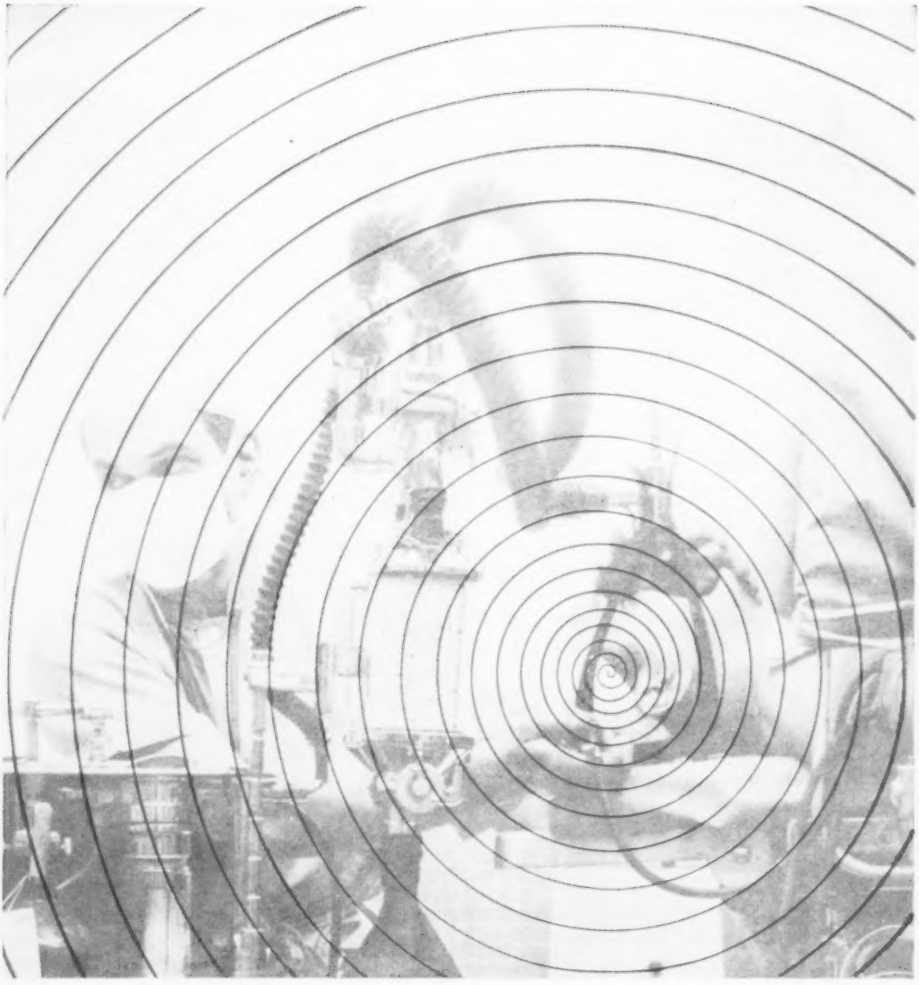

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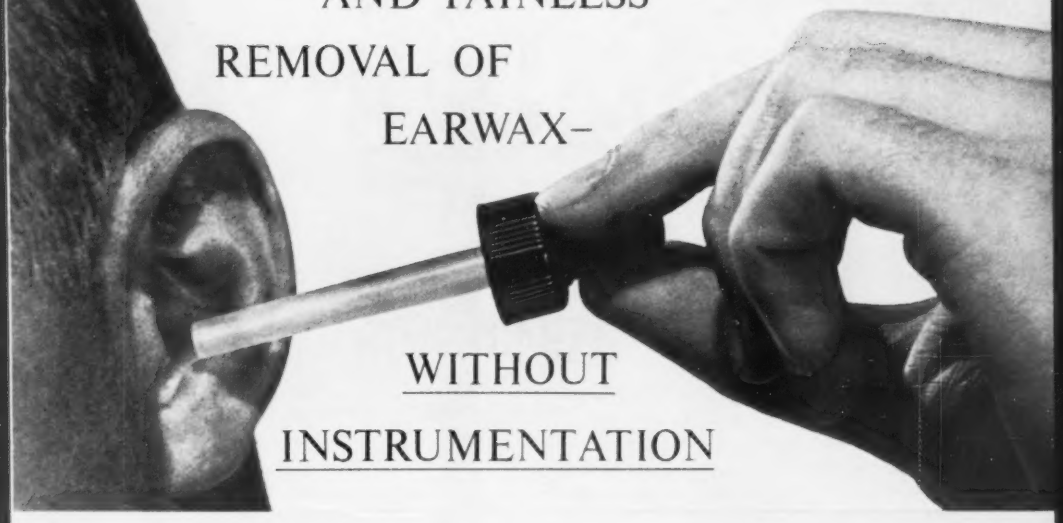

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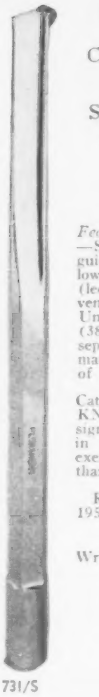

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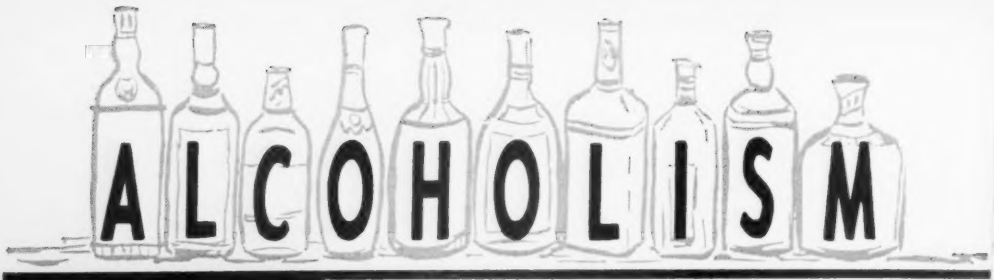

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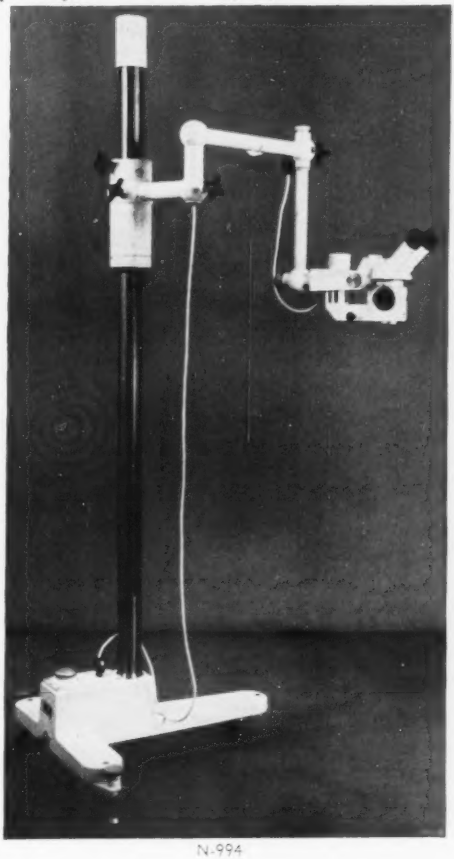

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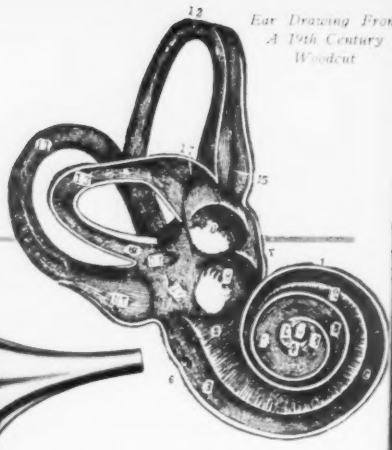

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Skin Diving

Its Physiological and Otolaryngological Aspects

J. ALLAN FIELDS, M.D., Fort Lauderdale, Fla.

Definition

Skin diving, as the term is now used, refers to the underwater sport of free diving, in which the participant wears a face mask and rubber flippers. Free diving implies that the diver is unencumbered by air or life lines extending to the surface. The sport has several ramifications, among which are spearfishing, photography, and exploration. In general skin divers are amateurs; however, there are a few professionals who teach the sport and market the various items of equipment. Skin divers are divided into two groups: (1) those who carry a self-contained air or oxygen supply, and (2) those who depend on breath holding. The former are able to carry out deeper and more extensive dives, the duration of which is limited by the capacity of the gas supply. The latter, of necessity, must be content with shallow dives of short duration.

History

Prior to World War II skin divers numbered in the hundreds; now there are several millions throughout the world. Apparently the almost sudden increment in the number of persons interested in the sport, was occasioned by the dispersion and subsequent return of service men from all parts of the world. For a number of years there has existed a nucleus of skin divers in the Pacific islands and the Mediterranean coast. As no equipment had been manufactured up to that time, each diver made his own, which, to say the least, was very crude. The main item consisted of a pair of lenses cemented into a hand-carved shell of wood, which was closely fitted to the orbital margins and held in place by an elastic band around the head. As proper fitting was extremely difficult, binocular vision was rare. Diplopia was most troublesome.[1] Japanese pearl and kelp divers fashioned a mask which covered both eyes and nose. This eliminated the diplopia. Except for a few modifications, it is essentially the same as that used today. By 1940, rubber fins or flippers were introduced, thus increasing the speed of the underwater swimmer tremendously and at the same time leaving his hands free. Various types of spears powered by rubber bands or springs came into being. With these simple pieces of equipment the skin diver came into his own. However, one further difficulty existed, namely, the diver was still harnessed to the surface by his frequent need to return for air. The Italian Navy had used a breathing unit to equip its underwater demolition teams. Some gear of this type is still in use, but it is not particularly suited to the skin diver's needs. The final impetus came in 1948 with the

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Presented as a candidate's thesis in partial fulfillment of the requirements for membership in the American Laryngological, Rhinological and Otological Society, Inc.

invention of the "Aqua Lung" by Cousteau and Gagnan.[2] This apparatus used compressed air, supplied automatically, at any given practical depth. The mechanical and physical principles of this unit, with only slight modifications, are used at the present time.

Burns[3] states: "Diving diseases and accidents formerly were very uncommon and understandably unknown to most physicians except for a few who were specially trained to cope with them in the United States Naval service. With the ever-increasing hordes of skin divers each year, diving accidents are becoming more commonplace. In California from January, 1953, through April, 1955, there has been an average of about one fatality per month. There were, of course, many nonfatal accidents, and there is obviously a need of improved safety programs for the diving public. Most urgent also is the need for the medical profession to be brought up to date in this field."

Physiology

The medical mishaps which befall the skin diver have, for the most part, been previously encountered by the commercial suit diver. They involve the air and water pressures to which the diver is subjected in his underwater environment. As far as actual pressure applied to the body is concerned, it makes no difference whether a suit is worn or the skin is subjected directly to the water pressure.

Boyle's law states that if the temperature remains constant, the volume of a gas varies inversely with the pressure.[4] Thus, the greater the pressure to which the gas is subjected, the smaller the resulting volume. Atmospheric pressure at sea level varies within small limits and is insignificant from the diving physiology standpoint. The weight of our atmosphere, at sea level, exerts a pressure of 14.7 lb. per square inch and is called one atmosphere. An additional atmosphere is exerted for each 33 ft. of depth below the water surface. Sea water is slightly heavier than fresh water but that

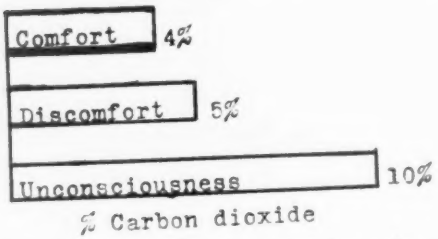

Fig. 1.—Volume reduction, diving time, and rate of volume changes between the various depth levels frequented by the skin diver.

is of little importance to this discussion. Since descending 33 ft. from the surface has doubled the pressure, the volume of gas has been reduced to one-half. At a depth of 66 ft. an additional atmosphere is added, with the resultant volume one-third of the original sea level volume. It is apparent, then, that, as the depth increases, the volume of gas is progressively smaller, but the change in the original volume varies by a smaller percentage (Fig. 1). For example, from the surface to 33 ft., gas loses 50% of its volume; conversely, there is a gain of 50% on returning; yet there has been a change of only one atmosphere. A change of one atmosphere between 198 and 231 ft. results in a gas volume change of only 2.5% of the original volume. From the foregoing, it can be seen that the first atmosphere or two below the surface is of the greatest concern to the skin diver, as this zone reflects the greatest changes in gas volume. The importance of these fluctuations in gas volume at shallow depths will be shown subsequently.

Dalton's law, the law of partial pressures, states that in a mixture of gases, each gas exerts its own pressure independently of all the other contained gases, and that the total pressure of the gas mixture is the sum of the pressures of all the constituent gases. The solubility of the various gases comprising air in the body fluids is directly proportional to the partial pressure exerted by each.[4] Of most concern to us are oxygen and nitrogen. Oxygen constitutes approximately 20% of air, but it is the only part of inspired air that is of use to the body.

Nitrogen makes up approximately 79% and is inert; however, at increased pressures nitrogen dissolves in the body tissues, especially fat. During rapid decrease of pressure it is released from the tissues faster than it can be carried by the blood to the lungs and released. Intravascular bubbles are thus formed, which in turn block capillaries and result in anoxia of the various tissues supplied. The symptoms resulting from this are known as caisson disease, "the bends," or decompression sickness.

Equipment

For a further understanding of the skin diver's physiology, it is necessary to describe briefly some of his equipment. The face mask is the most essential item and consists of an oval rubber shell fitted with a glass plate at one end and feathered, soft edges at the other, curved in such a way as to fit the upper facial contour closely. The nose and eyes are enclosed within the mask, the mouth being outside. Since there is entrapped air in the mask, a normal cornea-air interface is maintained, and there is very little change in the visual image; however, there is some magnification of size and distance.

The snorkel, which takes its name from the German submarine, is a short "J"-shaped plastic or rubber breathing tube which allows the diver, while on the surface, to keep his face in the water, swimming along until he spots his quarry below. It is used by the diver who relies on breath holding while making short dives.

For deep or sustained dives a self-contained underwater breathing apparatus (SCUBA) is used. The simplest and most popular type consists of a flask of compressed air (70 cu. ft. at 2200 p. s. i.) strapped to the diver's back. The air is supplied through a demand regulator, by a tube to the mouth, at the exact pressure of the sea water at a given depth. This is accomplished by the slight negative pressure of inspiration activating a diaphragm which is further compressed by the water pressure to which it is exposed. In this manner a set of levers bleeds the compressed air from the flask through a high-pressure valve. The expired air is exhausted through a separate tube. This is the open-circuit SCUBA. The closed-circuit apparatus takes into account that the body uses only about one-fifth of the oxygen inspired and wastes the other four-fifths. By removing the carbon dioxide of the expired air this oxygen can be rebreathed. In practice this is accomplished by breathing into a bellows, to which is connected a cannister of soda lime to remove the carbon dioxide. A pure oxygen flask with a needle valve replaces the volume lost.

A few divers use masks and helmets supplied by air from surface pumps and compressors. The limitations of the hose and the disadvantage of needing a tender make this method unacceptable to most divers. Their greatest use is in salvage work when latitude of movement is not so important.

There are several limitations and dangers inherent in each of the above-described types of gear. In the open-circuit SCUBA the greatest drawback is the limited air supply. As shown by Boyle's law, the addition of each atmosphere of pressure further decreases the volume of air available. Dives to three and four atmospheres (66 to 99 ft.) are quite common in skin diving. At these depths the volume of air available is reduced to one-third and one-fourth the initial surface volume respectively. The commonest size flask used contains 70 cu. ft. at 2200 lb. p. s. i. pressure, a supply sufficient for about an hour if used near the surface. Reduction of this volume to one-third or one-fourth limits the supply to the diver, to about 20 and 15 minutes respectively (Fig. 11). Air refills cost about $1.50; thus the price of the dive varies directly with the depth. This, however, can be looked upon as a veiled blessing since the diver is forced to return to the surface, having in general little chance of contracting the "bends" by so short a stay. There is a dangerous, growing practice among amateur divers of carrying along extra flasks of air, returning

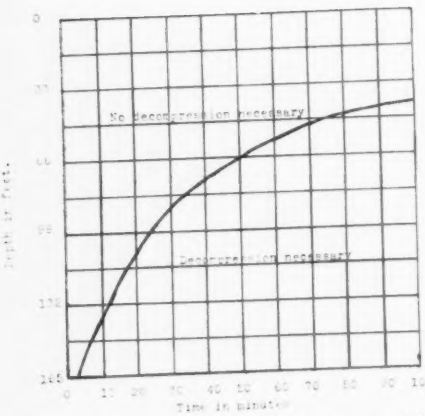

Fig. 2.—Safe limits of depth and time in which the diver need not decompress.

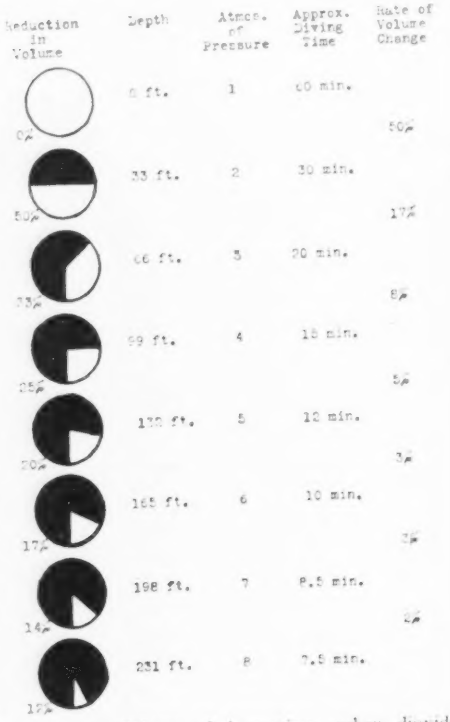

Fig. 3.—Effects of increasing carbon dioxide levels.

to the surface, making an air change and redescending. The nitrogen absorption is additive, and multiple dives within one 24-hour period increase the danger of bubble formation if decompression is not practiced, and a skin diver is loathe to waste his air for decompressing (Fig. 2).

Equipment failure in the open-circuit SCUBA is very rare. Its operation is automatic. Very little attention is necessary other than thorough cleansing after use. The closed-circuit or recirculation equipment has several disadvantages which render it less popular with the sport diver. Constant manual valving is necessary with depth changes to replace the changing oxygen volume in the breathing bag. The hazard of increased carbon dioxide concentration is ever present. This is usually caused by worn-out, inactive, soda lime. The average person at sea level breathes approximately 1 cu. ft. of air per minute, of which the lungs extract one-fifth of the available oxygen, giving up to replace it an equal volume of carbon dioxide. Expired air, then, is composed of carbon dioxide 4%, oxygen 16%, and nitrogen 79%. Concentrations of carbon dioxide above 5% make breathing uncomfortable, and at 10% unconsciousness supervenes. Cold, fear, and increased exercise cause the concentration to rise. Then, as far as carbon dioxide is concerned, the margin between comfort and symptoms is only 1% (Fig. 3). Should water leak into the soda lime cannister, a caustic, alkaline solution is formed in the air lines which could be quite dangerous.

Breathing pure oxygen is safe at depths no greater than 33 ft. Further increase in depth may produce symptoms of oxygen poisoning.[2,3,5] Oxygen poisoning depends on the pressure of the oxygen being breathed. The partial pressure of pure oxygen is five times that of the oxygen contained in the air at the same depth. When pure oxygen is used, 66 ft. is the critical depth and between 33 and 66 ft. there is a possibility of symptoms. These are tingling sensations, irritability, nausea, muscle twitching, and, finally, convulsions. A suit diver, breathing air at depths in excess of 300 ft. is liable to have difficulty, as at this depth the partial pressure of

oxygen has risen to a sufficient level to make its influence felt.

Another drawback to the rebreather apparatus from the skin diver's point of view is that the equipment does not lend itself conveniently to depth changes. Proceeding from one level to another requires the diver to be constantly occupied with adjusting the needle valve to the new volume which his breathing bag has assumed owing to the increase or decrease of water pressure at the new level. This annoyance is so great that this fact alone has relegated the apparatus to almost obscurity in sport diving, notwithstanding the quietness of respiration, permissibility of longer dives, and relatively low cost of operation.

Dangers

To the uninitiated the thought of strapping on skin-diving gear for the purposes of exploring the bottom of the ocean or for fishing seems foolhardy. It is true that the sport does offer a few real dangers. Most of the difficulties encountered by the skin diver are those of his own making. The dangers of paramount importance encountered are those of equipment failure, water pressure environment, and panic of the diver himself. The prevalent conception that the diver is laying himself open to attack by monsters of the deep is, for the most part, without foundation.[2] There are recorded instances in which sport divers have been attacked by fish, but these are rare.

In south Florida, during the past four years, there have been three instances of fatalities among skin divers. None was a proved case of equipment failure. One case involved an attempt at a record-breaking dive. As the diver was being traced by depth-recording instruments it was noted that after he had reached a depth between 200 and 250 ft., he began to descend rather rapidly and continued to do so until electronic contact was lost. The body was never recovered and the cause of this fatality has not been determined. It is the thought of some that this was a case of nitrogen narcosis in which the diver did not recognize the symptoms and failed to institute proper means for its relief, namely, returning to the surface. The second was one of drowning after the air supply had been exhausted. This should not be considered as an equipment failure. The cause of the third death has remained a mystery, as the diver was discovered in approximately 10 ft. of water. No evidence of equipment failure was discovered in this case.

Almost every diver has experienced the annoyances of abrasions and cuts caused by contact with sharp coral. Numerous pricks by the long-spined sea urchin, which are rather painful at the time of infliction of the wound, are not serious, however, and amount to no more than the slight degree of infection which follows. There have also been numerous intense but local skin reactions caused by contact with the so-called "fire coral." This is a plant, resembling staghorn, which grows on coral reefs. The sting of the jelly fish and contact with the tentacles of the Portuguese man-of-war excite rather uncomfortable local skin reactions but have no serious import. However, a death caused by a "man-of-war" was recorded in a Navy Medical Newsletter of 1944. This involved a swimmer, not a skin diver. As far as can be determined, there was no other medical report made of this instance. Although the so-called sea lice bite or sting does initiate intense itching, and sometimes urticaria, these are, for the most part, not serious unless they involve the ear canal and concha. It is doubtful if this sting is the direct cause of the external otitis which follows; however, there is such an intense itching set up that the patient is almost compelled to engage in vigorous scratching of the part, making the reaction much severer than it would initially have been.

Although decompression sickness or caisson disease probably represents the single greatest hazard to the commercial diver, the danger to the sport diver is much less by comparison. The average skin diver uses a

single tank apparatus filled with compressed air which is equivalent at sea level to 70 cu. ft. of uncompressed air. This supply is sufficient for approximately one hour of normal breathing at very shallow depths. As the depth increases, as has been shown previously, this available supply of air, as far as the diving time is concerned, gradually decreases as the water pressure increases, following Boyle's law. At 33 ft. another atmosphere has been added and the diver's available air supply has been cut in half, so that he now has only 30 minutes of available air. Because of this limited air supply, caisson disease rarely occurs in the depths to which the skin diver descends (Fig. 1).

The greater the depth, the shorter the time required for tissue saturation by nitrogen. A quick dive and return may be made to more than 200 ft. with little danger of intravascular bubble formation. So short a time is insufficient for tissue saturation. There is an ever-increasing practice growing among skin divers to use multiple tank blocks or to make multiple tank changes during a given period of diving. Also the diver is growing bolder and, in many cases, careless. There is a natural tendency to explore greater depths as one loses his original restraint and prudence. This means more time on the bottom and more likelihood of bubble formation (Fig. 2).

Decompression sickness has a number of manifestations, all of which are due to the tissue anoxia caused by the accumulation of intravascular bubbles. Asceptic bone necrosis has not been reported as having occurred in skin divers up to the present time, although the possibility exists. Permanent paralysis, "chokes," Ménière's syndrome with its syncope, faintness, vertigo, scotomata, and diplopia have been observed numerous times in caisson workers and deep-sea divers. Decompression sickness is common in high altitude aviators.[6] Kinsey[7] reported two cases of central nervous system damage during submarine escape practice at depths of 100 and 50 ft. respectively. These accidents could easily happen to skin divers.

Nitrogen narcosis has been commonly experienced by both skin and deep-sea divers. This is a little understood form of intoxication which may lead the subject to irrational acts. It is a euphoria similar to that due to alcohol but minus the "hangover."[2] That this narcosis is due to nitrogen has been shown by the substitution of helium or hydrogen as the inert element of the breathing mixture of gases.[5,8]

The ability to think clearly and act accordingly prevents panic. Panic is a serious hazard to a diver. The absence of it, when in difficulty, has saved many a diver's life and the presence of it in a companion diver has endangered the lives of others. There are many new experiences encountered in this fascinating sport. Some are momentarily frightening and could lead to panic. In this state one is liable to perform irrational acts, for instance, removing the mask or intake mouthpiece. This then leaves him almost blind in the first instance and without air supply in the second. Immediate return to the surface is imperative. This can easily be accomplished by the diver who remembers to exhale as he ascends, but the diver in panic may close his glottis. Breath holding while ascending is probably the cause of all cases of air embolism. It is the most serious situation encountered in skin diving and often leads to sudden death. During ascent the reexpanding air is not allowed to escape through the closed glottis; pulmonary elements rupture, and there is massive air escape into the circulation. This accident has occurred in water as shallow as 10 ft.! The treatment is immediate recompression.

Carbon monoxide poisoning is another threat to the skin diver's safety. The inclusion of exhaust fumes in the compressed air supply, by improper isolation of the intake of air to the compressor and the exhaust of the gasoline motor, can reach harmful proportions. The use of electrically driven compressors eliminates this hazard. The symptoms, signs, and treatment of this condition are well known.

Otolaryngological Aspects

Of all the problems faced by the skin diver, the commonest are those falling within the otolaryngological field. For the most part, these are not serious.[9]

Since the use of most SCUBA necessitates breathing through the mouth, the neophyte skin diver is often faced with mild or moderate degrees of a condition known as "face squeeze." The nose, being covered by the face mask, is isolated from the remainder of the respiratory passages when the palate closes the nasopharynx. This prevents free pressure exchange between the mask and air supply. As the depth increases, the mask is forced tighter against the face, since the volume of air within the mask is diminished inversely as the outside water pressure increases. This difficulty is ordinarily taken care of by exhaling slightly through the nose in order to compensate for the loss of air volume within the mask. At times it is observed that while one is breathing with the nasopharynx open, there appears to be a slight negative pressure exerted in the nasopharynx and nose during inspiration. This removes a small portion of the entrapped air in the mask, creating a relatively lower pressure in the mask than outside. The combination of the water pressure on the mask and that over the face in general tends to squeeze the face into the mask itself. The tissues of the face become turgescent and dusky in color, and appear distorted. After such an occurrence it is not uncommon to notice a small amount of blood seeping into the mask. This is apparently caused by capillary ruptures within the nose and sinuses. During and following "face squeeze" there has been noted on occasions a rather mild headache, which, however, subsides rather rapidly on equalization of the pressure. This also is believed to account for some of the cases of severe ear pain. There being a direct unobstructed air passage between the relatively negative pressure in the mask and the orifices of the Eustachian tubes probably explains this mechanism. It is thought that the Eustachian orifices and the tissues within the Eustachian mouths themselves probably become turgescent in a manner similar to that which the face has been observed to assume. Should this progress to a degree sufficient to prevent the forcing of air into the ears, and further diving be pursued, possibility of rupture of the tympanic membrane becomes likely.

This ear pain associated with "face squeeze" is not to be confused with pressure sensations which immediately precede the need for clearing the ears. The persistence of this air lock due to Eustachian tube mouth congestion has led on several occasions to hemotympanum, the mechanism of which is thought to be that of turgescence of the vessels and swelling of the mucosal lining of the middle ear, with consequent rupture of vessels and exudation of blood into the space itself. This represents the counterpart of the aerotitis which is observed in aviation. This same mechanism may also lead, in a few days, to chronic Eustachian obstruction with ensuing serous otitis media. The treatment of these last-mentioned conditions differs in no way from the treatment of aerotitis which is commonly seen by otolaryngologists.

Another rather annoying, but ludicrous situation, is occasionally felt by the skin diver, namely, that of compression of the stomach bubble. It is to be remembered that as the diver descends and ascends he is in vertical orientation. However, on descent, being upside down, the stomach contents have reoriented themselves so that the cardioesophageal junction is bathed in gastric secretion. As the compression of the entrapped gas of the intestines, particularly if this should be a rather larger amount than usual, is combined with compression of the stomach bubble, often the contents of the stomach are forced into the esophagus and a small amount is regurgitated. Minute amounts may be aspirated and induce a rather annoying cough. This cough is in no way serious, but it does make breathing through the SCUBA rather uncomfortable. Occasionally following this

hiccup has been experienced, which in itself is most annoying, as by this mechanism a rather rapid "face squeeze" can be introduced. The mild headache and face pain which the diver often encounters, sometimes lasting for an hour or so, is thought to be due to the difference in air pressure between the more or less isolated nose, nasopharynx, and sinus air communications, from those of the remainder of the respiratory tract. These pains would probably not ensue if the entrapped air in the nose, nasopharynx, and sinuses was as susceptible to compression as the remainder of the body tissues.

The commonest complaints for which the skin diver seeks the aid of an otolaryngologist are those referred to the ear. Behnke [10] states, "Although air pressure has been increased to values as high as 225 pounds per square inch in sinal and aural spaces without demonstrable effect, small differences in pressure on the two sides of the tympanic membrane incident to tubal blockage of the order of 1 to 2 pounds per square inch (50 to 100 mm. Hg.) produce striking objective changes in the mucosa of the tympanum." The relative negative pressure within the middle ear and mastoid cells produces a "cupping action," the degree of which depends on the difference in pressure on the two sides of the drum head. Following retraction of the tympanic membrane, there rapidly appears a dilation of the vessels around the handle of the malleus, pars flaccida, and anterior and posterior plica. Pain appears shortly. Further increase in pressure causes rupture of vessels, releasing blood and serous fluid. The end-result is perforation of the tympanic membrane. Lambertsen [11] states: "Owing to a passive rise of all body fluids which corresponds to the increase in water pressure on the body, the blood vessels of the tissues of the middle ear will have a pressure within them even greater than the water pressure on the outside of the tympanum, by an amount equal to the blood pressure. Acute congestion of the tissues of the middle ear and tympanic membrane results. Capillary rupture may occur, resulting in the formation of blood bullae. Later fluid or blood may exude into the middle ear. If pain is ignored and the pressure increased tympanic rupture may occur." A similar mechanism causes sinus pain and bleeding if there is obstruction to free air interchange.

It has been well documented that the commercial diver should not practice his profession during attacks of Eustachian catarrh, colds, allergy, or upper respiratory infections. During these attacks Navy divers are disqualified for the period of these difficulties. The skin diver who has not been so well prepared in the possible disruption of physiology of the ear, nose, and throat as has the commercial diver, is frequently known to disregard these minor ailments in his enthusiasm to participate in the diving at hand. Most tympanic perforations occur before the second atmosphere of pressure is reached. During descent, which in all cases should be moderately slow, at about the depth of 10 ft. a small amount of pressure is felt in the ears. Most frequently this is felt simultaneously on both sides. This is the signal to swallow, make yawning movements, or press the face mask up against the external nares and exhale through the nose, thereby performing the Valsalva maneuver. If the diver, at this level, is unable to completely clear his ears, he should return to the surface and immediately give up diving for the remainder of that period. If rupture occurs, there is an immediate rush of sea water into the tympanic cavity. The possibility of carrying foreign material into the middle ear is enhanced considerably, particularly if there are plugs of wax or other debris in the external canal. Some degree of acute otitis media follows almost all of these underwater perforations. They are generally treated by adequate antibiotic coverage and frequent removal of discharge and debris by the suction method. Drops or other medication instilled into the canals should be avoided. Of the several underwater

tympanic perforations which I have observed, none has failed to heal.

A case of extremely severe external otitis has been observed following several "sea lice" bites within the entrance to the canal and about the concha. There were a number of small discrete, reddish areas surmounted by a tiny blister. The discomfort and itching was intense. When first seen the reaction resembled that of herpes zoster oticus. However, none of the other accompanying symptoms were present, and the patient gave a history of having been skin diving the preceding day. There was complete canal closure with pain in the ear and in the temporomandibular joint and moderate trismus. Since the patient had other "sea lice" bites around the neck and face, the severe course of this external otitis was probably generated by these bites. It was thought that the use of cortisone preparations in this ear materially shortened the duration of the disease.

Although very little has been written concerning the traumatic problems of the ear of the skin diver, parallels can be drawn from the published work in caisson disease and in workers subjected to conditions of increased atmospheric pressure. Alt [12] reported, in 1896, three rather severe cases of labyrinthitis in caisson workers. Each of these workers was subjected to the atmospheric pressures frequented by skin divers and for a comparable time. One experienced severe pain in the ears, vertigo, and deafness. Another, after leaving the air lock, experienced the sudden onset of severe pain in the chest, collapse, and total deafness. The third had cyanosis, dyspnea, deafness, and marked vertigo. The tympanic membranes in none of these cases was ruptured. In 1909, Keays,[13] reporting on 3692 cases of caisson disease, mentioned that 113 cases of vertigo were noted, pain in the ear and hemorrhages with temporary deafness in 68, and Ménière's symptom-complex in 14. He noted two cases of apoplectiform deafness. No attempt was made in the series to state the pressure to which the diver was exposed or the duration of time. Vertigo and tinnitus commonly appear after the completion of exposures to increased pressure environments. Observations by Lester and Gomez [14] in 1898, performed in the caisson itself, revealed hearing to be affected to a greater degree by bone conduction than by air conduction. The hearing levels for air and bone conduction were reduced in proportion to the pressure experienced. They were of the opinion that the pressure of half an atmosphere was sufficient to cause retraction of the tympanic membrane and two atmospheres caused congestion of the malleolar plexus and in some cases displacement of the ossicular chain with resulting tinnitus. Vail [15] states: "The ear may be affected in two ways by the direct action of compressed air. One, there may be a more or less mild form of trauma when the damage is confined to the middle ear. Two, there may be trauma when, besides damage to the middle ear, injury is done to the internal ear." Damage to the cochlea and vestibule are due to intravascular bubble formation. However, Alexander, cited by Vail,[15] thought that the inner ear symptoms appearing in caisson workers were caused by hyperemia and small hemorrhages in the internal ear and that the symptoms might appear acutely after the injury or could at times have a chronic progressive course. I have experienced, and have observed in three others, periods of brief vertigo. Examination of the ears in all four instances revealed nothing abnormal except the presence of a plug of cerumen in two. The vertigo disappeared shortly after surfacing, and there was no subsequent difficulty encountered. Only one of these divers experienced pain preceding the vertigo. Two were wearing compressed air apparatus. The other two were diving from the surface by breath holding. It is postulated that the vertigo in each of these was nothing more than unusual displacement of the stapes in the oval window giving temporary vertiginous symptoms.

Five cases of ruptured tympanic membrane have been observed in the past four

years. Two of these were in the same diver, and occurred on the same afternoon. In each there was difficulty in clearing the ears and some pain was experienced, but in only one was the pain considered to be severe. The diver who had both drum heads ruptured in the same afternoon was examined within two hours of the accident. At that time there was little recognizable as a tympanic membrane in the left ear. The skin of the external auditory canal near the drum was quite edematous. There was a small posterior superior perforation, which, when the patient turned his head to the side, placing the ear in a dependent position, exuded a rather large amount of serous fluid tinged with blood. The other ear exhibited nothing more than a traumatic perforation in the posterior superior quadrant with bluish discoloration of the drum head and some curling of the edges of the perforation. Considerable thought was given as to why such a large amount of fluid would drain from an underwater perforation. However, it was finally concluded that probably the fluid had emanated from the middle ear and mastoid cavity and represented a slight amount of bleeding plus the salt water which had rushed into those cavities following the rupture of the tympanic membrane. All of the tympanic ruptures mentioned above suffered a moderate amount of otitis media; however, all healed. The first four healed within a few days. The left ear of the case of bilateral perforation, the ear containing the fluid, required approximately two weeks for complete healing. In each case large doses of a mixture of streptomycin and penicillin were used as the antibiotic coverage.

An unusual case of hearing loss and tinnitus occurred two years ago and up to the time of this writing has not been satisfactorily explained. A shallow dive to approximately 20 ft. was made with no difficulty in clearing the ears. While the diver was sitting on the bottom, about five minutes from the beginning of the dive, a moderate pain was experienced in the left ear. This was not considered severe, and attempts at clearing the ears were made; however, the pain persisted. When he reached the surface, it had not disappeared. In approximately 20 to 30 minutes there was no discomfort. Numerous Valsalva maneuvers failed to produce any noise of escaping air through a possible rupture. Within an hour the drum head was examined otoscopically, and no perforation was found. Within the next day or two it was noted that a mild tinnitus of rather high pitch was present in the left ear. This was in no way annoying. There was no subjective hearing loss at any time. During a routine audiometric check, several months later, it was discovered that there was a hearing loss most pronounced in the 4096 frequency with a deficit of 40 db. At the time of this writing the hearing deficit has persisted. The tinnitis is also present, but has not reached an annoying level. The possibility of bubble formation within the labyrinth has been ruled out since the dive was to no more than 20 ft., and the duration of the dive at the time of experiencing pain had only been approximately five minutes.

Summary

Skin diving is a fascinating, rapidly growing sport. It suddenly boomed following World War II when the widely dispersed service men returned bringing with them items of equipment and knowledge picked up in the South Pacific and Mediterranean areas. There are now close to 5,000,000 amateur divers in the United States. With so many participants there is an acute need for an understanding of the medical problems involved. Though of a minor nature, most of these problems fall within the field of otolaryngology.

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Frontal Sinusitis

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This is a preliminary survey, in clinical perspective, of 200 external operations for frontal sinusitis carried out between 1927 and the present date, with illustrations from recent cases.

Cases tended to come before one whenever infections were prevalent; it was the aftermath of an epidemic of Asian influenza which prompted this contribution. After such sequelae there was generally a long gap before the next batch of cases. The annual incidence in the West Riding is about 7 per 100,000 of the community.

After the introduction of chemotherapy and antibiotics, fewer cases called for operation and more survived. This influence would be greater were it not for predisposing factors in etiology which could generally be discerned, consisting either of anatomic obstruction of the infundibulum by developmental deviations or pathologic blockage by subjacent polypoid degeneration.

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Infection was manifestly air-borne rather than blood-borne. In recent years, sensitivity to antibiotics was studied in every case. Experience proved that when abscesses formed there was no substitute for surgical drainage, and that permanent recovery depended on acquisition or reestablishment of active immunity.

In children, infection was often fulminating, with orbital invasion. In only five cases was I not called in until there was no perception of light in the affected eye. There were two or three in which an abscess had been incised and acute inflammation had subsided; permanent fistulae had formed.

In adults, x-rays sometimes revealed a fluid level which moved in a lively manner—with a meniscus concave upwards—on tilting the head from side to side; in these acute or recurrent "catarrhal" infections operation was withheld, pending a quiet interval, when intranasal or transantral procedures always sufficed. In about half the cases nasal polypi were present. There were many coincidences in man and wife and in families

Fig. 1.—In frontal sinusitis the more concave-upwards the meniscus of fluid exudates and the livelier the movement on tilting the head from side to side, the better the prospect of spontaneous resolution. A subsequent intranasal or transantral procedure is indicated.

Fig. 2.—Frontal sinusitis in a girl aged 11, with orbital abscess, suitable for paranasal rhinotomy.

—even among relatives residing in different continents. Some sufferers from polypi were conscious of disseminating common colds among contacts not previously so susceptible to them.

In old age, especially when simple polyposis had passed imperceptibly into papillary carcinoma, pyoceles of the frontal sinus were sometimes found: these were sterile or in a state of subacute inflammation. In complicated cases it was generally found expedient to remove the ethmoid cells and to drain the sphenoidal and maxillary sinuses.

Differential diagnosis rarely presented much difficulty. In childhood, dacryocystitis sometimes gave a superficial resemblance to the swelling of the upper eyelid characteristic of frontal sinusitis. Orbital cellulitis from pustular lesions produced more complete fixation of the eyeball, as though it were set in plaster of Paris, whereas in frontal sinusitis the diplopia was manifest only in upward directions of the gaze. Herpes zoster and septic thrombophlebitis of the cavernous sinus were among diseases which accounted for the fact that general practitioners referred many cases to ophthalmic colleagues. In one case, frontal sinusitis occurred a few days after the onset of typhoid fever during pregnancy, and other complications followed too swiftly to allow of surgical treatment; the patient died. Tumors and cysts in the vicinity, though of great variety, never simulated sinusitis.

In 80% of cases which came to operation paranasal rhinotomy was carried out. The radius of a quadrantic incision along the medial margin of the orbit was a little longer than that of the course of the angular vein: a communicating venule at the root of the nose was usually divided. The periosteum was incised. The insertion of the tendo oculi and the pulley of the superior oblique were reflected laterally, together with the orbital fascia and its contents, taking the utmost care that there should be neither protrusion of the orbital fat into the wound nor undue pressure upon the eyeball. The

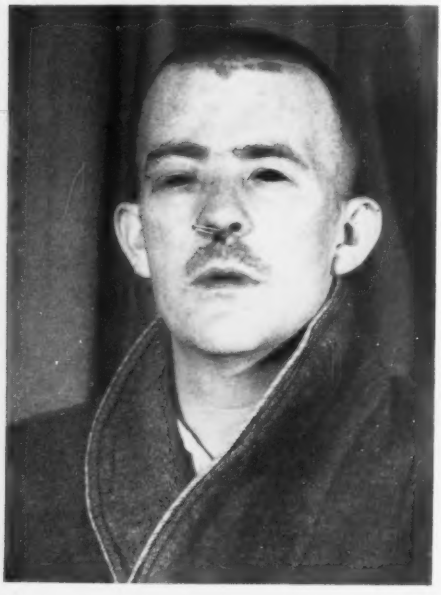
Fig. 3.—Woman 27 years old under endotracheal anesthesia preparatory to paranasal rhinotomy. Orbital invasion is manifest on the left side.

Fig. 4.—Man aged 64 undergoing resection of the ascending process of the superior maxilla during paranasal rhinotomy.

anterior and posterior ethmoidal foramina, with the vessels and nerves which emerged from them, marked the level of the cribriform plate within the cranium. The optic foramina were sedulously avoided. Unless a breach in the medial wall of the orbit had already provided a starting point for excavation, the infundibulum was opened just above the lacrimal groove. A sufficient extent of the floor of the sinus was removed to afford adequate drainage and surgical access. Only long continuous strips of ribbon gauze were introduced into the wound, and these were never cut, in controlling any slight hemorrhage; this was because a persistent sinus would immediately raise the possibility of a retained swab. In adjusting the new opening into the sinuses, Citelli's and Luc's forceps were invaluable. Whilst the ethmoid cells were being removed and the sphenoidal sinus was being opened, the possibility of abnormalities in the course of the optic nerve and ophthalmic vessels was borne in mind. Everything was done under brilliant illumination from the headlamp beam in a cavity kept dry by swabbing and suction. Part of the dense mucoperiosteal lining of the nose was used as a plastic flap to restore in some measure the mucosa of the infundibulum.

Fig. 6.—Girl aged 17 displaying healed scar six weeks after operation for frontal osteomyelitis.

Fig. 5.—Man aged 37 five days after operation for frontal osteomyelitis: healing by first intention of the coronal incision within the hair line.



Permanent patency of the infundibulum clearly depended on removal, medially, of a protuberance at the junction of the frontal bone and nasal septum and, anterolaterally, of an adequate portion of the ascending process of the superior maxilla. In one case where these steps were shown by x-rays, in the case of a war pensioner, to have been omitted, a ninth operation to close a recurrent fistula was successful.

Under such circumstances as lead to operation, the sinuses on the opposite side were seldom free from infection. Usually it seemed prudent to "let sleeping dogs lie." Sometimes a transantral operation upon the other side was carried out. When bilateral sinusitis was recognized at the onset, a median vertical incision between the angular veins was made, as described in a former communication.

Pansinusitis was commoner than a solitary mucocele or empyema, so it was often necessary to establish free communication between

Fig. 7.—The patient shown in Figure 6 six months after operation.

all the sinuses, by carefully adjusted enlargement of the natural ostia, to which all ciliary movement is directed. The turbinals were subjected to injury as little as possible, and the sphenoethmoidal recess avoided altogether.

Finally, to prevent postoperative hematoma, the cavity was packed with antibiotic gauze, arranged for withdrawal from the nostril, preferably under brief narcosis, in 36 hours. A drainage tube was also inserted. The wound was sutured in such a manner as to bring broad surfaces into apposition with meticulous approximation of the dermal margins. The temporary suture of the eyelids, sometimes inserted at the outset of the operation was removed if necessary and a "cracker" dressing applied.

Sometimes an empyema of the frontal sinus gained outlet into the orbit and simultaneously stripped off the frontal pericranium, simulating periostitis. The duration of the disease helped in the differential diagnosis; the briefer the history the better the prospect of satisfactory treatment by periorbital rhinotomy.

In about 20 cases, abnormal pneumatization of the temporal bone, with isolated cavities, predisposed to osteomyelitis of the frontal bone. This development brought about an appearance in skiagrams reminiscent of the pattern to be seen in polished marble or old red sandstone. Under these conditions, rather than rely upon continued antibiotics and small incisions, I usually carried out the following more extensive operation.

A coronal incision in the form of a Cupid's bow, with two convexities posteriorly meeting in the sagittal plane, was made down to bone within the hair line. The scalp and frontal integuments were retracted downwards. The anterior wall of the frontal sinus was removed. Infected parts of the frontal bone were excised, exposing the dura where necessary. In a number of instances, extradural abscesses were evacuated. The infundibulum was widened as in periorbital rhinotomy, admittedly as a life-saving measure, which might not give permanent security against recurrence, because of the essential difficulty of doing it thoroughly, from above. The wound was then closed in layers and drained through one nostril.

The surgical obliteration of the frontal sinus thus accomplished resulted in asymmetry of facial contour, but in the absence of adherent scars, this could readily be disguised. A subsequent transantral opera-

Fig. 8.—Healing paranasal scar in a 3-year-old boy, 10 days after operation.

tion, after an interval for convalescence, must be regarded as a normal concomitant of this operation. In one recent instance, the patient had to be readmitted for paranasal rhinotomy about a month later; this demonstrated the overwhelming advantage of the paranasal procedure at an earlier stage of the disease.

Good recoveries were the rule. There was a death, in addition to one mentioned above, from osteomyelitis of the frontal bone; this was before the introduction of antibiotics. Seven patients who had incipient meningitis or symptoms of space occupying lesions on admission were transferred to neurosurgical departments. Epileptiform attacks and signs of bacterial invasion in specimens of cerebrospinal fluid, cautiously withdrawn, were informative physical signs not masked by penicillin. Thus uncomplicated frontal sinusitis carried no mortality.

Five instances of blindness of the eye on the affected side were demonstrated before operation; sometimes this could only be done with difficulty, owing to swelling of the upper eyelid. Blindness of the opposite eye occurred in an arteriosclerotic subject owing to coincidental central retinal venous thrombosis; such possibilities are worth bearing in mind, when advising operation.

In the absence of recurrent nasal polypi, a good prognosis was uniformly vindicated.

Summary and Conclusions

To sum up, I have tried to portray against a clinical background a disease which affects about 7 per 100,000 of the community. Its onset is fulminating in some cases and insidious in others. It always constitutes a danger to vision and a threat to survival.

I trust that I have maintained, first, the overwhelming necessity which still prevails, when antibiotics fail, for timely intervention, of which the risks are slight and the outcome is almost uniformly good—for fatalities and permanent complications only occur when operation is performed too late—secondly, that paranasal rhinotomy in the manner formerly practiced by Chiari and others and so lucidly described by Mr. Walter Howarth is the operation of choice, even at the appearance of fluctuating frontal swellings; thirdly, that in bilateral cases a modification described by me in 1938 is suitable, consisting of a vertical incision between the angular veins; and fourthly, that in cases of osteomyelitis a coronal incision within the hair line gives a good exposure.

Mr. Howarth is to be congratulated for crystallizing out from so many concoctions a technique which stands so well the test of time. One axis of the coherent whole might be inscribed "provide for drainage," another "avoid mutilation," and the third "forestall complications." It applies old methods in a new way and seems destined to remain the operation of choice until such time as pyogenic diseases can be prevented.

Green Leas, Savile Park.

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Toxoplasmosis and Congenital Deafness

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Toxoplasma gondii, a protozoan, since its discovery by Nicolle and Manceaux, in Africa, and by Splendore in Brazil, both in 1908, has gained rapidly in reputation as the causative agent of a specific disease. Toxoplasmosis is now recognized in its ubiquitous, "pantropic" occurrence. A rapidly snowballing amount of related publications testifies to a universal interest.

This concern is understandable in the case of a disease which spreads all over the globe. In a normal urban population its incidence has been found to be 30% to 60%, on the basis of reaction to Toxoplasma antigen. Even more alarming data were given for certain groups of patients with other diseases. In mental patients rates based on positive toxoplasmin skin tests, sufficiently high dye test, and complement-fixation titer were as high as 50%, rising to 75% in Mongoloid idiocy. These incidence figures climbed even higher with age.

Many cases of advanced toxoplasmosis in the newborn became known, making antenatal infection probable. The mother acquired the disease during pregnancy or was already a carrier at the time she entered the pregnancy and transmitted the disease to the progeny. The intrauterine embryopathy, then, results frequently in stillbirth or prematurity, although the mothers generally present no symptoms of the disease. Transplacental infections occur through maternal leukocytes, with or without lesions to the placenta itself.

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Congenitally infected children show pathology most often limited to the central nervous system. Eye lesions are very characteristic, but many other organs may be involved.

Reports on participation of the ears are extremely rare. Callahan, Russell, and Smith (1946) reported the first case in which the parasite has been identified in histological sections of the mastoid process. A boy 6 weeks of age underwent the operation of bilateral mastoidectomy with the diagnosis of acute purulent mastoiditis. Death occurred 20 days later, and the pathological diagnosis was toxoplasmic encephalomyelitis, internal hydrocephalus, toxoplasmic bilateral otitis media, generalized icterus, hemorrhage between dura mater and calvarium. "The middle ears with a large part of temporal bone were sectioned following adequate decalcification. Examination of a section taken from the left ear disclosed a large amount of highly vascular granulation tissue in the operative wound that was infiltrated with a large number of polymorphonuclear leukocytes. There was liquefaction of the marrow with a marked destruction of the bony trabeculae in the mastoid part of the temporal bone that remained. Examination of sections taken from the right ear disclosed essentially the same type of pathologic changes as was noted in the left ear. Small intra- and extracellular microorganisms identifiable as Toxoplasma were seen in the sections from the left middle ear." The method of spread to the mastoid process was not clear, but the writer considered it unlikely that the ear served as entry.

Campbell and Clifton (1950) found adult toxoplasmosis in a family, with three members showing a more or less severe

degree of nerve deafness. Frenkel and Friedlander (1951) in a case of toxoplasmosis (No. 7.) described a wandering type of nystagmus, otitis media, and pharyngitis. Koch, Schorn, and Ule (1951) reported that intracerebral calcifications seen at autopsy induced the clinician to suspect toxoplasmosis; mucous-suppurative otitis media with mastoiditis was present on both sides. Dietzel (1958) found in serial examinations of patients with presence of the parasite or with positive dye reaction that toxoplasmosis has to be considered in the etiology of retrolabyrinthine hearing loss and nystagmus, emphasizing, however, the desirability of a framework in history and in concomitant clinical symptoms when the suspicion should be reinforced.

The ages of the patients were respectively as follows: 6 weeks (Callahan, Russell, and Smith); 19, 24, and 49 years (Campbell and Clifton); 5 years and 8 months (Frenkel and Friedlander); 3 weeks (Koch, Schorn, and Ule).

Considering the scarcity of related observations, the following reports may claim interest.

Report of Cases

Case 1.—The patient was first admitted to the Children's Medical Center Dec. 22, 1956, at 2 weeks of age because of drowsiness and anorexia. One week prior to admission the infant was seen as an outpatient because of twitching of the lower extremities and hypertension. During the week prior to admission the infant had been lethargic. Progressive lethargy and hypothermia was the cause of referral to the hospital.

The baby was the product of an uncomplicated pregnancy and delivery. The duration of labor was nine hours, with spontaneous respiration ensuing. Mild jaundice was noted at 24 hours and cleared spontaneously. Feedings were taken poorly. Birth weight was 5 lb. 13 oz. (2636.50 gm.), and at the time of discharge from the maternity hospital the infant weighed 5 lb. 10 oz. (2551.45 gm.)

Both parents were in their 20's and well physically and mentally. One sibling 1 year of age is alive and well.

Physical examination at admission revealed a depressed white female infant with an obviously enlarged head and exhibiting Cheyne-Stokes respiration. The head circumference measured 36.75 cm. The anterior fontanel was approximately 8×7 cm., and the sutures were markedly separated and bulging. Both pupils were irregular and fixed, and black lacy material was seen anterior to the retina at 2 o'clock. Respirations appeared shallow and irregular. Heart rate was 60 to 50 per minute. The abdomen was moderately protuberant with enlargement of liver and spleen. Neurological examination revealed a lethargic, hypothermic infant (T. 89.8 F). The grasp reflex was apparently adequate, while the suck reflex was poor. All extremities were movable actively.

Laboratory data on admission revealed hemoglobin 16.5 gm. %. WBC was 3700; polymorphonuclears 40%, lymphocytes 60%. Lumbar puncture and bilateral subdural taps revealed xanthochromic fluid. The ventricular tap was negative. Analysis of CSF revealed a 4+ Pandy; cellular elements totaled 60, with 8 mononuclears and 52 RBC. Skull x-ray revealed a large head with no definite calcific deposits.

The infant was placed in an isolette and 150 cc. of 5% dextrose solution in water commenced. The temperature improved, as did the hydration.

A ventriculogram revealed definite hydrocephalus and a suggestion of some soft tissue or fluid-filled masses in the inferior frontal area. A block was noted in the region of the foramen of Monro or third ventricle. Moreover, injection of P. S. P. in lateral ventricle was not present in the spinal canal 15-20 minutes later.

On Dec. 26, 1956, the head circumference was 38 cm. Ophthalmologic consultation revealed bilateral chorioretinitis, moderately severe. On Dec. 27 Toxoplasma organisms were seen in the ventricular fluid, by Dr. Thomas H. Weller. Subsequently the infant became cyanotic while being fed, and further ventricular tap yielded 20 cc. of xanthochromic fluid from the left ventricle. Total protein in CSF was 960 mg. %, RBC 200, and WBC 10. On Dec. 28 the abdomen became distended, although no free fluid was present in the peritoneal space. The head size was decreasing (36.3) although left-sided convulsions occurred. On Jan. 2, 1957, the infant suddenly died.

The gross pathological diagnosis was as follows: congenital toxoplasmosis; calcification of cerebral cortex; internal hydrocephalus; hepatomegaly; splenomegaly; calcification of renal cortex; interstitial pneumonia; pulmonary edema; hilar lymphadenopathy; solitary cyst of liver.

In the brain the cerebral hemispheres were grossly collapsed and deformed (Fig. 1). Scattered irregular patches of a firm yellowish-gray material, presumably calcium, were present in the subpial layers. The ventricular system was tremendously dilated except for the temporal lobes. The aqueduct was well visualized and appeared patent. Histologically, the lesions within the brain were extensive, of a granulomatous type. These granu-

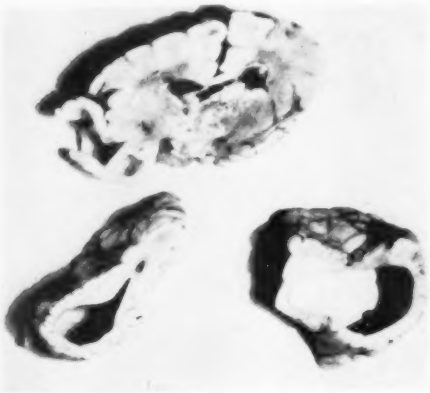

Fig. 1 (Case 1).—Macroscopic aspect of the brain.

lomas were located within the cortex and, to a less degree, within the gray matter and brain stem. The granuloma contained central necrosis and calcification surrounded by round-cell infiltration and gliosis. Perivascular cuffing was moderate, and many eosinophils were present. Both pseudocysts and vegetative forms were observed throughout the brain.

Complement fixation titer in the maternal blood was 1:64 and the titer in the infant's blood was doubtful. (This is understandable in view of the poor antibody production in infants under 90 days of age.) An unusual feature was presented by the fact that organisms could not be isolated, particularly after staining both in the vegetative and the cyst forms, from the brain and cerebrospinal fluid. A sign of immaturity was offered in the immature marrow.

Ears.—The external ears were normal with the external canals probe patent. The left tympanic membrane was intact, but a small crescent area of hemorrhage was present in the inferior half. No infection or other gross pathology was present in the left middle ear.

The right ear was removed in toto and, through the courtesy of Dr. Sydney Farber, Professor of Pathology, was released for further examination. It was prepared in the usual way with celloidin embedding; sectioning was done in the horizontal plane, yielding 750 sections, which were stained with hematoxylin-eosin, Heidenhain-Mallory, some auxiliary stains like Gomori impregnation, and examined in full and polarized light and with phase contrast.

The histological work was done by Miss Dorothy Linden and photomicrographs were prepared by Mr. Donald C. Withee.

The outstanding features of this series can be summarized as follows:

The osseous walls of the middle and the internal ear were found in adequate state of development. Ossicles and labyrinthine capsule were found in a condition in accord with the age. The sensory end-organs were fully developed and normal. Calcium deposits were found in the spiral ligament, localized predominantly in the vascular stria. The dura was intact, without engorgement.

The tympanic membrane showed a deep retraction without perforation (Fig. 2). Directed against the lower part of the promontory, the deep sac was filled with the desquamative products of the external canal.

The entire epitympanum was filled with mesenchyme and transudate, with the three ossicles solidly embedded (Fig. 3). The relation of mesenchyme and inflammatory product was a complex one. The mucous membrane lining of the tympanic cavity showed considerable thickening and in spots cellular infiltration, but no engorgement, no extravasates. On the other hand, innumerable foci of organization, in the form of resorption buds, pro-

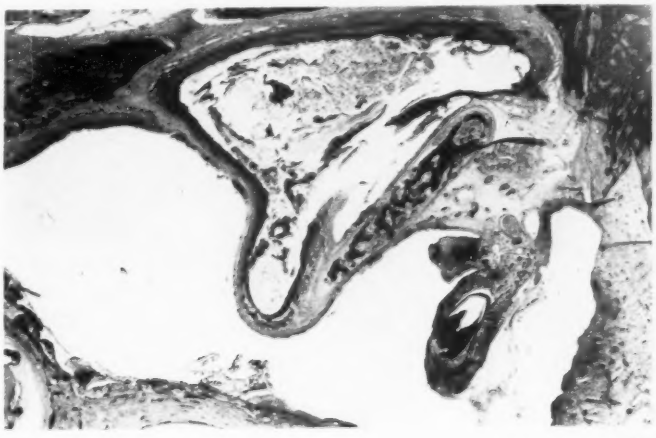

Fig. 2.—Deeply retracted tympanic membrane. Mallory; reduced about 36% from mag. ×16.

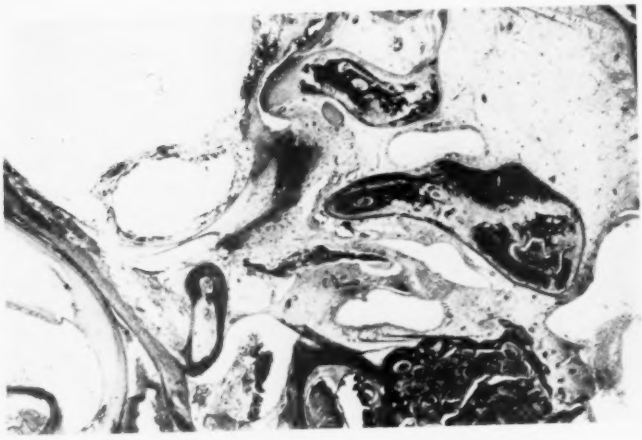

Fig. 3.—Epitympanum, ossicles embedded in mesenchyme and inflammatory products. Mallory; reduced about 36% from mag. × 16.

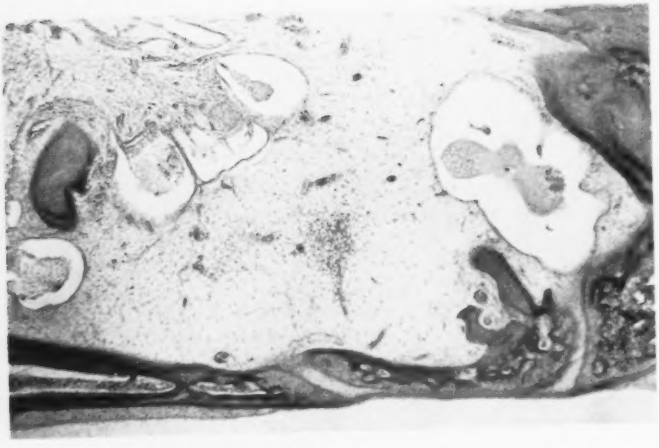

Fig. 4.—Hyaline organization in oval window; rearrangement buds. Hematoxylin-eosin; reduced about 36% from mag. × 85.

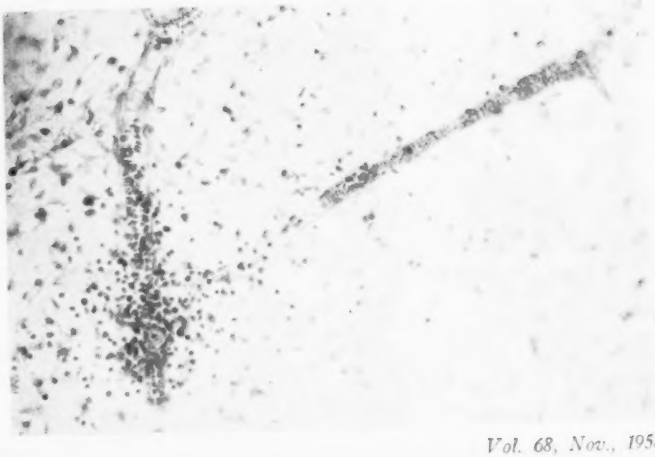

Fig. 5.—Perivascular infiltration in mesenchyme. Mallory; reduced about 38% from mag. × 250.

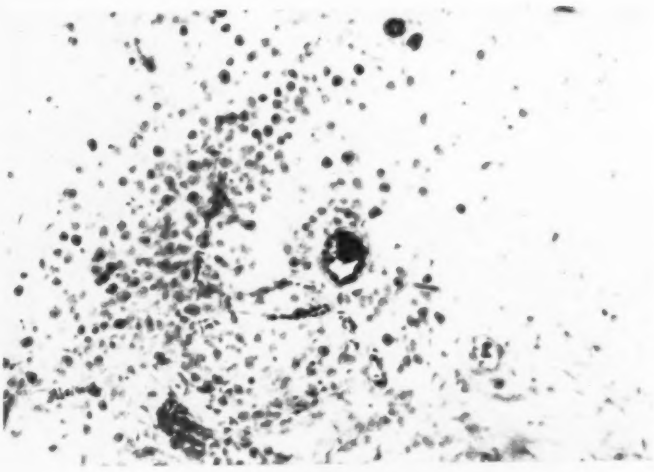

Fig. 6.—Perivascular infiltration in mesenchyme. Hematoxylin-eosin; reduced about 36% from mag. × 250.

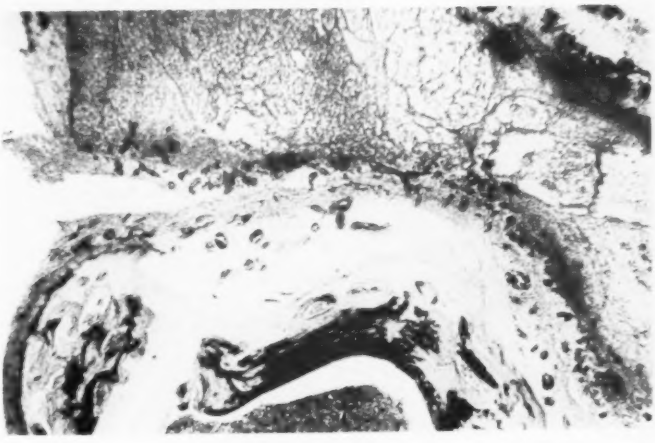

Fig. 7.—Fibrous organization around incudomalleolar articulation. Mallory; reduced about 38% from mag. × 85.

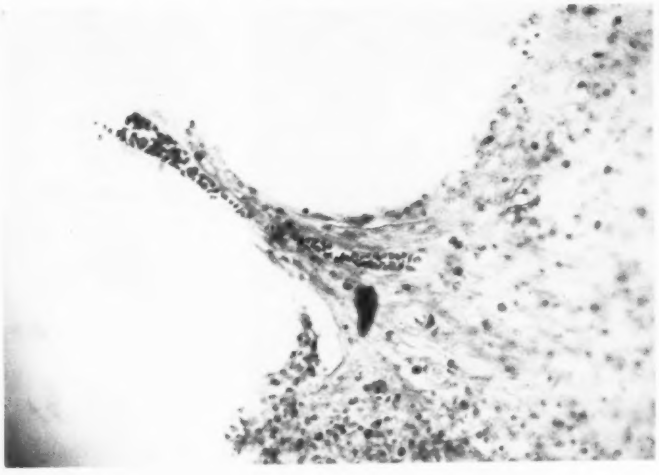

Fig. 8.—Torn rearrangement bud with central vessel. Mallory; reduced about 36% from mag. × 250.

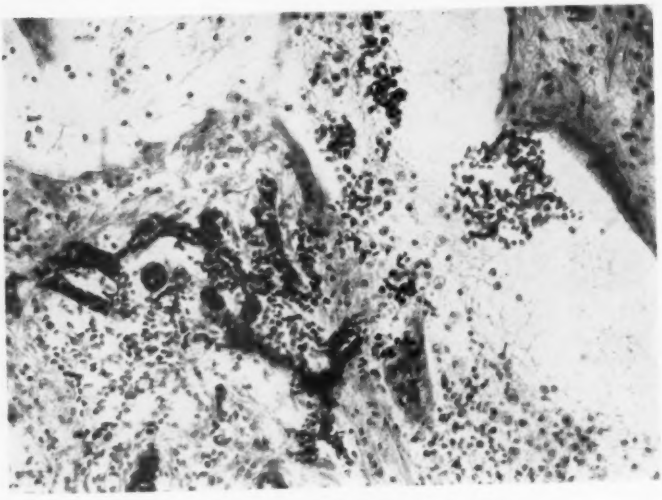

Fig. 9.—Blood pouring from torn rearrangement bud (right upper corner). Mallory; reduced about 36% from mag. ×250.

trated from the submucosa (Fig. 4), entering the masses which filled in the spaces between the cushions of the thick mucous membrane. These masses themselves were homogeneous but with islands of dense cellular infiltration around vessels (Figs. 5 and 6). But cellular infiltration and small fibrous patches were observed equally in perfectly homogeneous, avascular areas. Between the buds a row of pseudocysts was enclosed, giving the characteristic rosary-like appearance. At many points the surface of the mucous membrane was broken with outpouring of lymphocytes which then were caught in the transudate. This phenomenon was even more conspicuous where dense fibrin meshwork formed in extended areas of the filling material, as around the incudomalleolar articulation (Fig. 7), surrounding it like a plaster cast. Broken organization buds (Figs. 8 and 9) showed that they carried a central vessel serving as a root to the dense vascular network inside the secretion masses. While a network of coarse fibers was woven around the large ossicles, the niche of the oval window was seen filled by a hyaline mass; between the crura solid bands were formed. Within the more massive remnants of the mesenchyme, perivascular cuffs of lymphocytes were frequently seen.

The mesenchyme showed hyperemia, cysts, infiltration, and fibrinogenesis, and its arrangement was as follows (Fig. 10): nearest the tympanic wall one found a transparent, somewhat loose layer with interspersed fibroblasts and numerous vessels. In the next layer the vessels became sud-

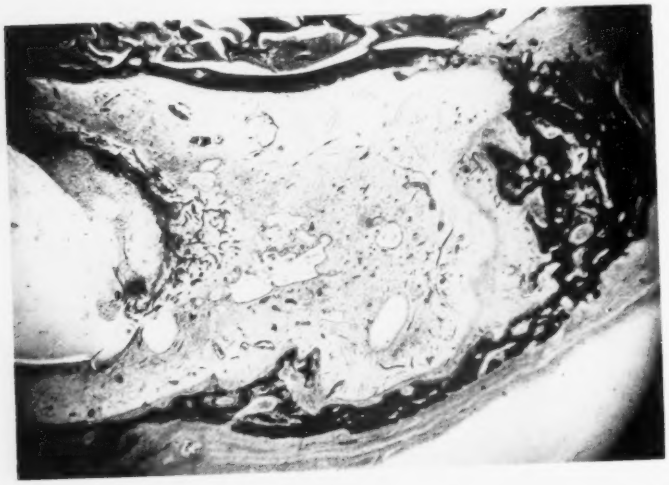

Fig. 10.—Structure of mesenchyme (details in text). Mallory; reduced about 36% from mag. ×32.

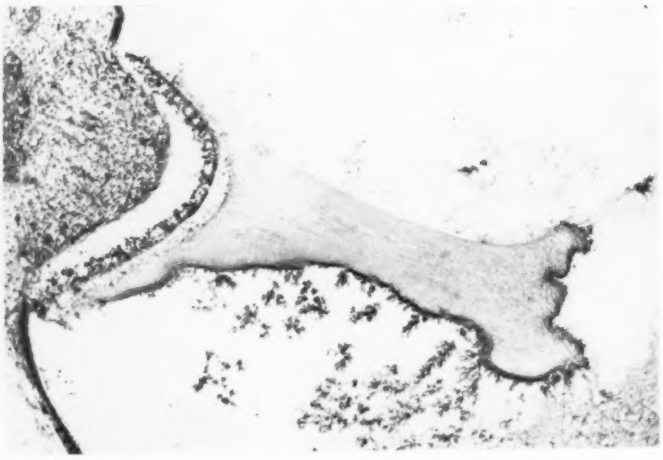

Fig. 11. — Cupula: one edge intact, other dissolving at contact with extravasate. Hematoxylin-eosin; reduced about 36% from mag. ×125.

dedly much wider and engorged, but the intercellular tissue remained loose; above this was a cystic layer with considerable cellular infiltration and overlaid by a very rich conglomerate of vessels embedded into a less densely infiltrated strip of tissue; inward of this wall densest infiltration formed a strong walling-off. Next followed a densely woven mesh of fibers, among which conspicuous bands were forming while the cellular infiltration diminished. Here seems to lie the boundary of the mesenchyme.

In the internal ear mesenchymal remnants were floating within the endolymphatic spaces of the vestibulum, especially within the semicircular canals, where they were differentiated with difficulty from a transudate which here showed a slightly hemorrhagic character. The cupulas presented a well-known picture (Fig. 11). The edge was intact, but where even a few red blood corpuscles touched it, rapid destruction was the consequence. In the cochlea, besides the scalae the ductus cochlearis, too, showed mesenchyme content; the organ of Corti was intact in all turns, and the membrane of Reissner was in a normal or slightly depressed position. The ganglionic spaces of the canal of Rosenthal were empty (Fig. 12), corresponding to the upper half of the middle turn and lower half of the apical turn. The octavus stem was lacking from the specimen. Calcium deposits were seen, in the spiral ligament of the basal and of the middle turn, in the shape of sharply circumscribed granules in the vascular stria or of more diffuse, partly perivascular infiltrations (Fig. 13). In some sections a seemingly calcified seam accompanied the spiral ligament along a certain stretch. The circumference of the granules was uneven, and they were divided from the inner edge of the stria by one or two cell layers.

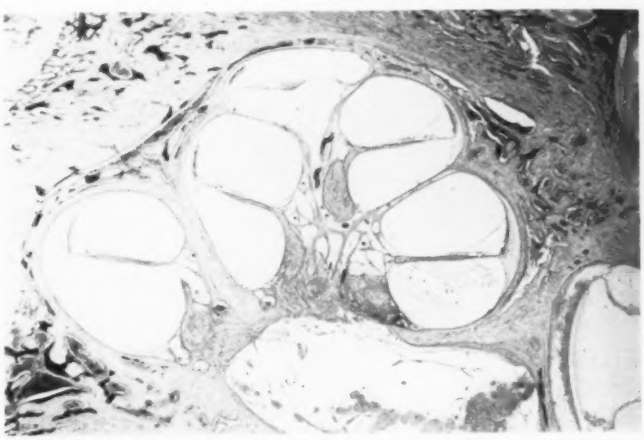

Fig. 12.—Axial cochlea —survey. Mallory; reduced about 36% from mag. ×7.

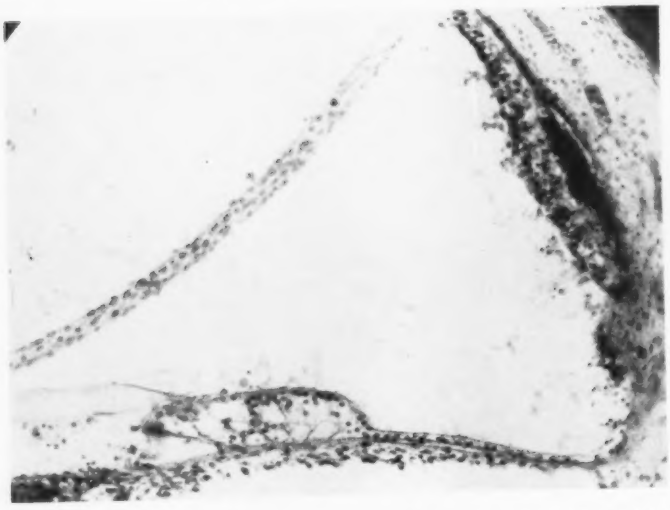

Fig. 13.—Calcification in the vascular stria of the middle turn. Hematoxylin-eosin; reduced about 36% from mag. × 225.

The picture added up to an otitis media with extensive organization most probably of intrauterine origin and showing, besides organized products, signs of an active inflammation.

Case 2 (Boston Lying-in Hospital).—This was a stillborn boy of 40 cm. crown-rump length and 3100 gm. weight. The course of the pregnancy was smooth, but in the seventh month of gestation the mother developed a maculopapular pustular rash over head, neck, and upper extremities, accompanied by severe headaches, fever, and cervical adenopathy. The disease was considered to be a streptococcic infection and was treated with tetracycline (Achromycin) and penicillin. There were five previous siblings: One was stillborn; another was jaundiced at birth, and a third died after pneumonia at the age of 7 weeks. The family was in contact with livestock and dogs.

A large hydrocephalic head was recognized by x-ray and, at delivery (Feb. 2, 1957) made perforation necessary, which was followed by podalic version and extraction.

At necropsy hydrocephalus was found (Fig. 14), and necrosis of liver and kidney. The notable pathohistological findings were increased hematopoiesis in the liver, much phagocytosis of hemosiderin pigment in the spleen, intraventricular and subependymal hemorrhage, in addition to the obviously dilated ventricular system.

It was recognized that hydrocephaly may have resulted from toxoplasmosis acquired in utero, especially in consideration of frequent contact of the family with animals. However, cultures from brain, kidney, and liver, inoculated into roller cultures, did not show evidence of cytoplasmic change. Diligent search for pseudocysts in all tissue sections demonstrated none.

Dr. Kurt Benirschke, pathologist of the Boston Lying-in Hospital, collaborated by releasing a hearing organ for further examination.

The right ear was examined, after embedding in celloidin, in a horizontal sectional series (technical details as in Case 1).

The dura was thickened everywhere. In the middle ear, mesenchyme was present in numerous locations, but what was left were only scattered fragments. Within these mesenchymal masses there was hyperemia with extravasates around the larger vessels; conspicuous blood lakes were surrounded by a thin wall which might have been formed by a condensation of mesenchyme fibers. Mesenchymal filling was conspicuous within the stapedial obturator foramen. Cuffing was present around the vessels of the mesenchyme and of the mucous membrane. The hypotympanic cells were still filled by mesenchyme showing cellular infiltration and high vascularization. In the round

Fig. 14 (Case 2).—Macroscopic aspect of the brain.

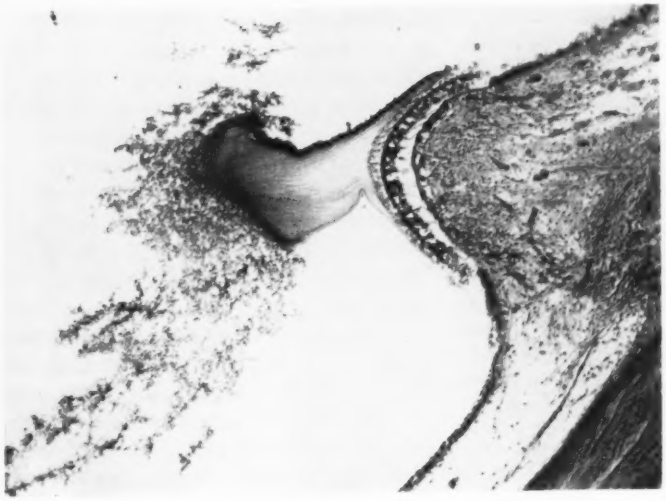

Fig. 15.—Cupula, in dissolution at contact with extravasate. Mallory; reduced about 36% from mag. ×125.

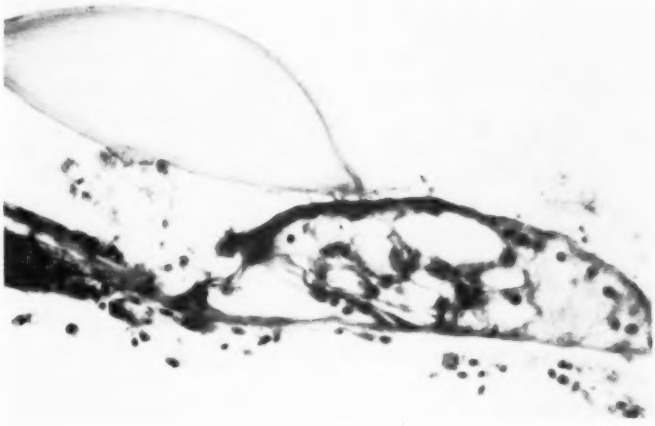

Fig. 16.—Infiltrated mesenchyme in cochlear duct. Mallory; reduced about 38% from mag. ×400.

Fig. 17.—Dilatation of cochlear aqueduct with endosteum crossing its orifice. Hematoxylin-eosin; reduced about 38% from mag. ×250.

window there was some blood which penetrated the membrana tympani secundaria to show up in the adjacent portion of the scala tympani of the basal turn (source of blood: the surgical intervention, perforation). The promontorial mucous membrane was engorged.

In the inner ear the crista superior was partially destroyed by a hemorrhage (Fig. 15); the organ of Corti was well preserved, except parts of its wall facing the internal sulcus (Fig. 16) (for a technical explanation of this phenomenon see Kelemen, 1956); the cochlear aqueduct was dilated with the endosteum apparently running over its cochlear orifice (Fig. 17); the internal acoustic meatus contained some blood.

Summing up, the premature ear (7 months) was remarkably free of mesenchyme for this age, and there were signs of an acute intrauterine inflammatory process. Blood may have originated from the perforation, but may point to a hemorrhagic character of the otitic process.

Comment

Toxoplasmosis was followed with increasing interest after Castellani (1914) for the first time attributed a death to this infestation. Wolf, Cowen, and Paige (1939) succeeded first in experimental transmission of human toxoplasmosis to animals; Pinkerton and Weinman (1940) published the first case seen in an adult.

The all-invading presence of the disease was only slowly appreciated. The percentage of infected population was given according to different titration values taken for positive. The titer of a population does not express manifest disease; in the overwhelming percentage of cases it lists only residua of a former infection. Positivity in the newborn means only transmission from the mother and no active disease. Two *pro mille* was given as the incidence of connatal lethal cases. The largest contingent was furnished by fetopathy, namely 142 cases (Essbach, 1957).

The protozoan parasite, of crescentic shape, 7μ in length and 2 to 4 in width, is Gram-negative and "one of the most ubiquitous and prevalent of parasites." Every warm-blooded animal can be infected with it. It is transmitted in the form of a low-grade infection to the offspring; it may be acquired during adult life and need not be fatal. From human lesions animals can be reinfected. Essbach stated that 0.1 cc. of centrifugal peritoneal exudate intravenously kills a healthy mouse in seconds. The routes of transmission are unknown except in congenital cases where leukocytes traversing the placenta are considered to carry the infection.

Decisive testimony regarding the presence of the disease is given only when the parasite is demonstrated in secretions or tissues. The organism has been isolated from tissues which did not show any other signs of toxoplasmosis. To authenticate cases by isolation of the organism, or at least visualizing it in the tissues, is a hard task. Blood, sputum, or liquor will yield it very rarely. So recourse has to be taken to serological tests. These are the cutaneous test with toxoplasmin (Frenkel), complement-fixation reaction (Warren-Sabin, Westphal), dye (methylene blue) test (Sabin-Feldman). High degrees of titer values can persist after healing. Although the parasite is found more readily in acute cases, it is very difficult to find in chronicity. Late cases may show the most characteristic pathologic lesions without giving the possibility of demonstrating the parasite itself. Antibodies develop quickly, and when this occurs the parasite leaves the blood and takes refuge in the tissues, initiating here the production of the pseudocyst, a most characteristic formation.

Propagation may proceed along nerves and blood vessels. Toxoplasma is an important pacemaker for other infections, above all, tuberculosis. No influence has been discovered of it as an agent causing malformation in the organogenetic period of differentiation, in contrast, e. g., to rubella.

Under the microscope the nerve cells may appear crowded with parasites. Perivascular cuffing is present. In 50% of the cases calcification was found which might reach degrees where detection by x-ray would be possible. Mummified terminal colonies,

pseudocysts of 20μ to 100μ in diameter, may form a refuge for the parasite or, as in the case of necrotic cysts, the point of its disintegration. "Sabin's triad" comprises hydrocephalus, cerebral calcification, and choroidoretinitis.

As was the case with many other pathologic conditions, exact descriptions of the histological picture existed long before one became aware of their connection with this particular parasite. Köhn (1952) emphasized that it is possible to identify cases of congenital encephalitis Virchow as being of toxoplasmic origin; many of the detailed descriptions of Schwartz (1924) in connection with birth trauma are recognizable as pseudocysts or calcifications. Toxoplasmosis as an important agent in encephalitis was only recently fully disclosed, but a group of pathologists (see Essbach) denies any connection between Virchow encephalitis and toxoplasmosis. The warning of Wohlwill (1921) should be remembered, according to which entirely different influences can produce quite similar processes in the infected brain; not the character of the lesion is of significance but the specific reaction of nervous tissue, especially when it is still in a state of development.

Clinically, many of the connatal cases, frequently prematurely born, show icterus. The manifestations are identical in all ages, differing as to acute and chronic, and variously located cases. With a course of one to eight weeks, a maximum near two years was observed. Milder forms are very common, but often not being characteristic they are enrolled under the names of grippe or gastroenteritis. The only instance where full suspicion of a possible toxoplasmic origin is awakened is stillbirth and prematurity.

The following will try to give a list of symptoms scattered in case histories which are in relation to the ear and the upper airways and postulate the interest of the otolaryngologist. In evaluation it is useful to keep in mind the possible results of congenital brain lesion.

Headache, convulsions, vomiting, vertigo, high temperature may be present. Acute lymphadenitis is accompanied by fever, while, after healing, indolent occipital, cervical nodes are left behind. Stomatitis, angina ulcerosa, sore throat, cough, rhinitis were mentioned, and a syndrome reminiscent of mononucleosis (lymphadenopathy, atypical lymphocytes). Facial paralysis may be present, toxic degeneration of cranial nerves. Hearing loss may go under the heading of osteomyelitic deafness, especially as the latter is known to be present, possibly, without osteomyelitis (Kelemen, 1957). Signs of infection through the nasal and oral mucous membranes follow, as encephalomyelitis with meningitis. Hydrocephalus presents a complication at delivery.

So far only the cases of Campbell and Clifton showed a "nerve deafness," described as mild, severe, and very severe, respectively, and increasing with time in two of the three cases.

Cysts (pseudocysts) were demonstrated in locations of otolaryngologic relevance in different reports: in the saliva, sputum, in the parotid glands, in the muscles of the floor of the oral cavity, in lymph nodes, in the esophagus.

Therapy has remained so far a discouraging chapter. Sabin (1950) explained that, although various drugs have been found capable of killing Toxoplasma in vitro, they were without effect in vivo. Essbach, seven years later, still stated that, even with massive application of every known drug, very abundant parasites can be demonstrated. As to chemotherapy, "Much labor and scanty yield characterize the results in this field" (Weinman, 1952).

All clinical experience regarding toxoplasmosis is based on three groups of cases. The first shows the parasites in secretions or the tissues. In the second, parasites are not demonstrated but serologic tests give positive readings. In the third, direct demonstration *and* serology remain negative, but the clinical history and, in case of autopsy, the microscopical findings permit identification of the case as toxoplasmosis.

Cases with visualized organisms being very rare, it is impossible rigidly to exclude the testimony yielded by the last group.

Among the instances discussed in this presentation, the first case has shown the parasite in abundance under the microscope; the second was clinically highly suspect of the disease, with adequate pathologic signs, but neither serology nor histology succeeded in definite identification.

In both cases, ossification was in adequate progress according to the age, and the changes were confined to the soft tissues. Their condition can be summarized as follows.

Mucoperiosteum.—Case 1: promontorial mucous membrane highly infiltrated; cylindric epithelium reaches, from the tubal corner, unusually high up; organization buds numerous; cells pouring from the submucosa are caught in the fibrous mesh of the mesenchyme. Case 2: extravasation in the mucous membrane of the tubal corner; engorged promontorial mucous membrane; perivascular cuffing within the obturator foramen.

Mesenchyme.—Case 1: epi- and mesotympanum filled; large ossicles embedded in mesenchyme, which is present in all endo- and perilymphatic spaces; foci of dense infiltration in many locations; cellular infiltration shown, among other locations, in the mesenchyme of the cochlear duct; fibrous organization around the incudomalleolar articulation; in the oval window a hyaline mass; vacuoles with transudate and with fibrinous content; around some groups of vessels, cuffing; from the mucoperiosteum cells pour out to be caught in the fibrous meshes inside vacuoles. Case 2: Spaces of the middle and the internal ear widely free of mesenchyme; remnants of the latter in the upper parts of the epitympanum and in hypotympanic cells; in the former location large blood lakes with definite wall formation and cuffing around larger vessels, extravasation; in the latter location, cellular infiltration and intense vascularization; in the niche of the oval window, cushion with high degree of vascularization, inclusive cuffing.

At this point it is of importance to remember that processi, buds of submucosal origin, can serve equally the process of organization or reabsorption; they are, generally speaking, the factors in rearrangement.

From the diagnostic as well as the prognostic viewpoint the status of the mesenchyme claims full attention. Structure and perseverance have to be observed, both being of decisive influence to the hearing function. The entire mesenchymatic development in the hearing organ is a process not strictly polarized but reaching the end-stage in an often devious manner, meaning repeated appearance and reappearance of identical stages. Guggenheim, Clements, and Schlesinger (1956), with full justification, called dissolution of the tympanic mass a most intriguing problem, with determination of the incidence of unresorbed mesenchyme in a general pediatric autopsy material a study long overdue. Rüedi (1937) explained that cushions disappear rapidly at any stage; on the other hand, similar mesenchymal structures can be built up quickly at any period when mesenchyme is present at all. Individual differences can be explained by the disposition and density of the fibers which for their part derive from different forms of fibrocytes.

The connective tissue cells, migrating cells (histiocytes), intercellular substance, and collagenous fibers, the four main components of mesenchyme filling the spaces of the middle ear are present in variable proportions. According to Schwarz (1931) all these disappear at the 10th month from the tympanic cavity and, normally, the pneumatization of the latter is terminated at this date. No pockets of mesenchyme were observed by Wolff (1934) in any specimen originating from 42 cases between the ages of 7½ fetal months and 72 years, beyond 13 months of age. She pointed out the way to distinguish between true mesenchyme and newly developed delicate areolar tissue, both types of tissue arising from the same ears

in which the mesenchyme has not yet been completely resorbed.

The core of the problem remains to hold apart the "original" mesenchyme from the products of inflammatory reaction which appear already in intrauterine life. Wolff offered the following formula: If the delicate stroma exhibits large full nuclei and is completely homogeneous with the submucosa at every level in which it appears, it may be considered as mesenchyme. If, on the other hand, at some levels it shows the surface of the mucosa extending through the tissue, this means that the young granulation tissue, or a polyp-like structure, has grown out from some irritated region and is encroaching upon the neighboring mucosa.

It is in the course of the transformation of the myxomatous tissue—which is fully developed, after a start around the third or fourth embryonic month, in the sixth or seventh month—into loose fibrillar tissue and, later, into the mucoperiosteum that combination with inflammatory products has to be watched. Formation of bands, the regular suspending apparatus of the ossicles, on the one hand, and pathological residua, on the other, are proceeding simultaneously. Precollagenous and, after transformation, loose or dense groups of fibrils show every kind of arrangement, and forebodings for the future have to be read from this complicated picture.

In the present cases the condensation of mesenchyme into definitive mucous membrane was not everywhere achieved. Conspicuous was the high degree of cellular infiltration in the mucous membrane. A phenomenon of interest was the emigration, the outpouring of white blood cells, through breaks in the surface epithelium at numerous points. The cells were then caught in the fibrous mesh of the mesenchyme, presenting foci of cellular infiltration.

The mesenchyme shows these foci at many points, in contact with the mucous membrane or far away from it. The arrangement of the mesenchymal mass has been described above. Where it recedes two kinds of vacuoles are formed. One is a roughly circular or elliptical opening, and its walls are without lining, resembling simple tears: the other kind is surrounded by a wall formed by compressed layers of fibers. Both are filled by mucous transudate, sometimes with fibrous content.

Besides this "fading away" of mesenchyme there is active resorption by buds originating in the submucosa and entering the masses scattered in the lumina of the tympanic cavity, remnants of mesenchyme and inflammatory products, and granulatory masses alike. These processi carry a central vessel, easily demonstrated "in situ" or by outpouring of blood when the processus is torn.

Fibrogenesis reaches a high degree within the mesenchyme. With impregnation it can be demonstrated that the fibers peel off the vessel walls. Already in this early stage they are condensed in bands and represent a serious threat to ossicular mobility, while blocking of the windows is achieved by near-hyalinized cushions.

The vascular system of the mesenchyme shows very different degrees of engorgement, as described above. An outstanding feature is perivascular cellular accumulation which appears around single vessels in form of cuffing and often surrounds a branching segment.

Personal observations have shown two ways in which mesenchyme disappears from the middle ear. In one instance, the vessels disappear first, leaving shreds of mesenchyme which are then resorbed piecemeal. In the other instance, the "parenchyme" of the mesenchyme disappears first and the vascular system persists, isolated, for a certain period, e. g., in the stapedial niche. Cause of the dissolution remains for both cases equally unknown.

As to congenital conditions which lead to hearing loss, the interest centers in the question of direct organization of mesenchyme remnants into cushions. This question is far from being fully elucidated. That the vascular net tarries longest in the window niches—here, too, systematic studies

are wanting—would seem to make this possible, in the presence of or even without an inflammatory factor. If this view is accepted, the much discussed question of foreign-body reaction in the etiology of newborn otitis (e. g., Schwarz) would lose much of its interest.

Fibrotic products need not be considered as irreversible. L. K. Guggenheim (1943) described the "reversible tympanic fibrosis" in children. An experimental approach to the problem of reversibility, by way of reinfection, has been tried (Kelemen, 1958).

As seen in the present cases, signs of irritation were numerous. Hyperemia was frequent, in both instances; the mucoperiosteum was not yet everywhere definitely differentiated, with still uncertain boundaries; cuffing, another sign of irritation, was common. Fiber formation from fine fibrils to coarse bundles—outside the loci of differentiation of the permanent ligaments—could be followed as starting from the vascular walls, the fibers peeling off from the outermost layers. Activity reached its acme in the niche of the oval window with engorgement, rich formation of vascular meshes, cuffing, and, even in this early stage, organization, which proceeded to hyalinization.

Together with the inflammatory signs in the mucoperiosteum, engorgement, and cellular infiltration, the entire picture cannot be disregarded in its potentialities. A phenomenon has to be emphasized as seemingly not observed hitherto: the outpouring of cells through the epithelium of the newly differentiated mucosa, to be caught in the fibrous meshes of the mesenchyme. It recalls the transmigration of cells through the cryptal epithelium of the tonsils. Here, as there, the meaning of this mass movement is not understood. The cells, after transmigration, obviously will be reabsorbed, with unknown effect of their constituents on the surroundings. To follow the parallel with the tonsils, a tropism might be considered originating there from the crypts, here from the mesenchymal mass. It is possible that more intensive studies will show this cell transmigration to be a constant activity around the final disposition of the mesenchyme.

As to the calcium deposits in the inner ear the report of Rollin (1934) can be cited. He found similar deposits, in analogous location in three cases: in two children, 2½ and 6 years of age, respectively, and in a man 59 years of age. He considered these deposits as of a dystrophic origin based on a local lesion of the tissues. Further discussion is left for a forthcoming publication of a case where similar deposits were found in a newborn of a mother with pseudohypoparathyroidism (Hinojosa, 1958). Regarding the present case, it should be recalled that calcium deposits were reported in the brain, in the subpial layers, in the form of granulomas within the cortex, where they were surrounded by round-cell infiltration.

Summary and Conclusions

Two cases of toxoplasmosis are added to the scant reports on aural pathology in the newborn in this condition. In the first case, the parasite was demonstrated; in the second, general pathological signs offered the basis to suspect toxoplasmosis. While in the ear the skeletal elements and the sensory end-organs were found to be in an adequate developmental stage, conspicuous changes were found in the soft tissues, mesenchyme, and mucoperiosteum. In the first case, with calcium deposits in the brain, similar formations showed up in the spiral ligament, predominantly in the vascular stria.

Analysis of these changes pointed to an irritative state running the whole scale from engorgement to organizatory products. Every sign reached its acme in the niche of the oval window.

Infantile toxoplasmosis is known to make a first-line attack on the central nervous system, and it must be claimed to be one of the hitherto undisclosed sources of congenital hearing defects.

Toxoplasmosis being present in an overwhelming segment of the population of all

climates, the elucidation of its role in congenital deafness asks for a large-scale assault on the problem at the pathological and the clinical level.

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Nonchromaffin Paraganglioma. Tympanic and Jugulare

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Nonchromaffin paragangliomas [1] as a group are histologically like the carotid-body tumors and are in essence blood-vessel tumors with interspersed epithelioid cells. I shall limit my remarks to those tumors which originate within the middle ear and the dome of the jugular bulb and I shall attempt to segregate them by their specific sites of origin to tympanic tumors of the middle ear [2] and glomus jugulare tumors arising from the jugular bulb. Published case reports are limited to approximately 100 cases. I feel that some tumors of this classification have been missed for lack of specific pathological identification. The objective of this study of a few cases in the Vancouver area is an attempt to make a special effort to emphasize early symptoms and early diagnosis.

Tumors originating from the nonchromaffin paraganglioma vary according to their particular point of origin, such as the tympanic plexus or the adventitia of the jugular bulb. These present either a slow-growing tumor or a more rapid invasive type of tumor. The pathological and microscopic pattern may vary from that which is very similar to a paraganglion structure to the potential hemorrhagic angiomatous type with a rich vascular network and a thin endothelial lining.

There is a variation [3] in nonchromaffin paragangliomas with abundant epithelioid cells with abundant cytoplasm, and the other type of tumor with a rich vascular network. This latter tumor may frequently present hemorrhagic tendencies within the tumor itself. The tumor is more likely invasive when the site of origin is in the adventitia in the dome of the jugular bulb rather than when it is confined to the middle ear, as on the promontory or invading locally into the Eustachian tube or oscillular chain and occasionally the labyrinth. Therefore, the histopathogenesis and site of origin of this tumor must be considered with the marked variability of symptoms, for when it is confined to the middle ear the symptoms are otological [4] and not particularly convinc-

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Fig. 1.—Early tympanic glomus tumor—promontory.

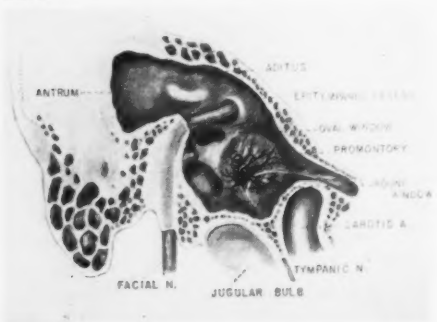

Fig. 2.—Glomus jugulare tumor—adventitia of the jugular bulb.

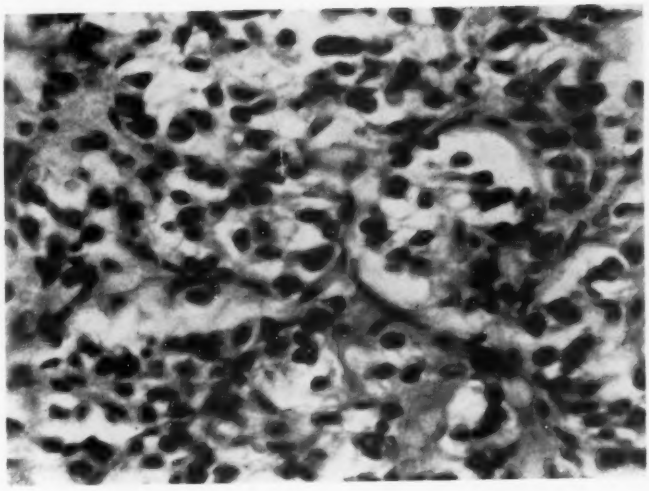

Fig. 3. — Microscopic section of nonchromaffin paraganglioma. Hematoxylin and eosin; × 425.

ing—for example, loss of hearing, tinnitus, aural discharge, vertigo.

An expanding middle-ear growth, while the tympanic membrane is still intact, may show some evidence of pressure on the tympanic membrane, with a reddish or bluish discoloration of the tympanic membrane with or without a noticeable pulsating tinnitus. In this particular case one must have glomus tumors in mind and be prepared to carefully plot a proper approach for an adequate biopsy. When the tumor has penetrated the drum membrane into the aural cavity it may present spontaneous hemorrhagic tendencies. If interfered with, as in the removal of a polypoidal or granulomatous growth in this area, biopsy is indicated. If it is necessary to remove the tumor by a radical mastoidectomy, then the bone work should be completed before attempting to remove the tumor, with a useful knowledge of handling almost any kind of hemorrhage which may occur. Adequate packing, including absorbable gelatin sponge (Gelfoam), epinephrine packs, electrocoagulation,[5] or even tying off the external carotid artery or internal jugular vein may be used. Perhaps this group with modifications includes 25%-40% of the tumors studied. The vast majority of these tumors when confined to the tympanic cavity can be removed surgically with meticulous dissection, using high-power loops.

On the other hand, the tumor which originates in the dome of the jugular bulb has possibly already involved the jugular bulb or eroded through the floor of the tympanic cavity and is in truth the glomus

Fig. 4.—Large tympanic glomus tumor—pressure on tympanic membrane.

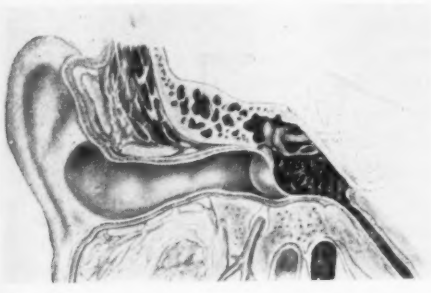

Fig. 5.—Tympanic tumor perforating tympanic membrane—presenting in aural canal.

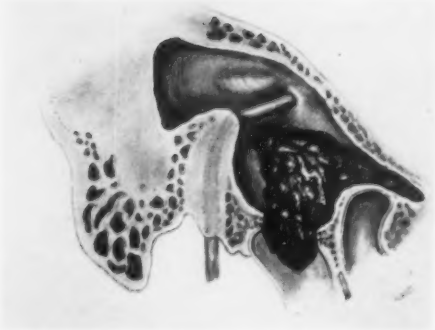

Fig. 6.—More advanced glomus jugulare tumor.

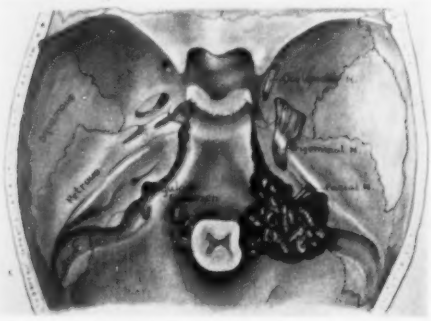

Fig. 7.—Expanding glomus jugulare tumor—petrous bone—compressing brain stem.

jugulare tumor, while the first discussed tumor confined to the middle ear is a tympanic glomus tumor. The real jugular tumor is more prone to invade bone and grow along the vascular network to the petrous tip and cause clinical vascular phenomena of major proportions. It may present neurological symptoms, the facial nerve appearing to be frequently affected. It may also affect any of the other cranial nerves from 5th to the 12th, depending on the manner in which the tumor is expanding. These cases are frequently first seen by the neurologist; sometimes a basal skull x-ray will show erosion which simplifies the diagnosis.

If the tumor cannot be removed with all osseous and soft tissue pathology included, the surgery can be followed by a thorough course of radiation.[6] Therefore the symptomatology from the tumor involving the area of the jugular bulb may be entirely neurological and vascular, and when well advanced the tumor may cause pressure on the brain stem. When all these morphological and histopathological factors are conjointly considered, the ideal treatment when possible is complete surgical removal of the tumor. In cases which involve the jugular bulb the hemorrhage[7] may be of such proportions that it is necessary to quickly pack and get out, treat the hemorrhage, transfuse the patient, and follow up with radiation therapy. I shall leave the radiosensitivity of these tumors to the radiologist.

In my study I am not overly enthusiastic about primary radiation except as an adjunct to surgery and in so-called nonsurgical cases, such as petrous erosion, recurrent tumors, and incomplete surgery. On this first point I differ with Graf[6] and Williams.[8] Nevertheless, I am of the opinion that the rate of growth of some of these tumors has been retarded by radiation, the tumors have sometimes been reduced in size and remained in this state for some years.

A thorough study of the clinical and pathological aspects of the tumor with the added knowledge of its particular tendency to recur, its potential to become malignant[9] at least locally, the necessity of carrying out thorough meticulous surgery with a follow-up of radiation and radium therapy becomes obvious. The diagnosis, because of the varied clinical gross appearance, is histological, making biopsy essential. Because of the variability in the rate of growth of these tumors, a long tedious follow-up is demanded, for some of them will recur whatever the therapy. Hemorrhage is a common feature, and cranial nerve involvement just as common. On examination 50% of the tumors can be seen presenting in the aural canal. If one is on the alert, a dry ear with a localized reddish or bluish discoloration and pressure on the eardrum and a so-called pulsating tinnitus with the above clinical symptoms requires complete investigation.

Report of Cases *

CASE 1.—A 44-year-old woman with aural fullness, tinnitus, and aural hemorrhage with intact tympanic membrane was first seen in October, 1951. Radical mastoidectomy was performed in November, 1951, and revealed a tympanic glomus tumor, which was completely excised. No recurrence occurred in six years.

CASE 2.—A 69-year-old woman reported with a continuous otorrhea and roaring tinnitus that had been present for five years, when a spontaneous facial paresis occurred. Following a radical mastoidectomy in 1945, recurrent hemorrhagic otorrhea became evident, requiring a revision of radical mastoidectomy in February, 1953, when, on account of severe hemorrhage, a vascular tumor was not completely removed. Radiation therapy failed to diminish size of growth, and paresis of the 10th, 11th, and 12th cranial nerves became apparent. Recent examination revealed no clinical change neurologically or increase in size of the tympanic tumor.

CASE 3.—A 43-year-old woman seen in May, 1955, presented aural fullness, pulsating tinnitus, and hearing loss of two years' duration. Examination revealed an intact tympanic membrane under pressure. A right radical mastoidectomy showed a tumor arising on the promontory and filling the inferior half of the tympanic cavity, which was carefully dissected. Recent examination presents no evidence of recurrence.

CASE 4.—A 62-year-old woman was first seen in December, 1954, by one of our neurologists. She presented a two-year history of hoarseness, pulsating tinnitus, a recent onset of vertigo with nausea, numbness of left arm and leg, atrophy (L) side of tongue, paresis of 8th, 9th, 10th, and 12th cranial nerves, and left vocal cord paresis. Biopsy of a tumor mass, base of skull near second cervical vertebra, revealed a nonchromaffin paraganglioma —glomus jugulare. X-rays showed a basal erosion with petrous apex erosion. Surgery was not considered. A course of radiation therapy followed. Recent examination showed no further clinical developments or radiological changes.

Case Study Analysis

Early Symptoms

(Variation due to vascular and epithelioid ratio)

1. Aural fullness (3)
2. Pulsating tinnitus (3)
3. Hemorrhagic tendency (2)
4. Auditory—hearing loss (3)
   (*a*)—otorrhea (1)
   (*b*)—vertigo (1)
5. Middle ear to aural tumors (1)
   Bleeding polypoidal to granulomatous (2)
6. Slow expansile growth (4)
   Benign may become locally malignant
7. Variation in gross appearance of tumor (4)
8. Frequent regrowth of tumor (1)
9. Commonly found in middle age females (2)
10. Spontaneous facial nerve paresis (1)

Clinical Findings

A. Middle ear—tympanic glomus tumor
   (*a*) intact tympanic membrane—early (2)
   (*b*) aural canal—later (1)
B. Glomus jugulare—less early (1)
   —spontaneous 7th nerve paresis etc.
   —petrous bone involvement with basal erosion
   —x-ray sometimes diagnostic
C. Combinations
   Site—middle ear—adventitia jugular bulb—adventitia jugular bulb—middle ear—petrous bone—brain stem

Treatment

Complete surgical excision Class A

Less likely Class B and combinations A and B

Combine with radiation therapy in selected cases owing to uncertainty of recurrences and inoperable states where petrous bone has been involved, etc.

Adequate preparation to treat hemorrhage and combat shock when necessary

Advanced angiomatous tumors, e. g., glomus jugulare, less amenable to surgery—radiate

Radiation may delay and slow up tumor rate of growth

Early diagnosis essential

Exacting follow-up of all P. O. cases

Three cases—radical mastoidectomy (1 case simultaneously radiated)

One case—radiation; nonsurgical

Three- to six-year follow-up; all females—average 54 years; all alive with no further recurrence

Two cases—tympanic glomus tumor with complete surgical excision to date

One case—glomus jugulare tumor with combined surgical excision (incomplete) and radiation therapy; clinical progress stationary at this time

One case—glomus jugulare tumor with neurological extensive involvement and petrous apex erosion; radiation therapy only; clinical progress stationary

Summary and Conclusions

Surgical excision should be carried out when possible and is particularly desirable in early cases.

When surgery is incomplete, combine with radiation.

* Nonchromaffin paraganglioma by biopsy.

When tumor tissue recurs, radiation is recommended.

Palliative radiation in neurological involvement with basal erosion may slow tumor rate of growth.

Early tympanic glomus tumors are amenable to surgery.

Glomus jugulare tumors are less amenable to surgery, particularly when expansile. Combine with radiation.

Early diagnosis is essential.

Repeated postoperative examination is recommended.

Drs. G. A. Badger, B. W. Tanton, and N. J. Blair gave permission for the use of their cases. Mrs. Nan Cheney, University of British Columbia, prepared the medical drawings. Miss Kay Hoskins, Vancouver General Hospital, prepared the photographic slides.

925 West Georgia.

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Serous Otitis Media in Children

A. NEIL LEMON, M.D., Philadelphia

During the period of 1947 to 1957 it was noted that many of the children referred for removal of tonsils and adenoids had moderate hearing problems. Some of these showed improvement in hearing after tonsillectomy and adenoidectomy, but others did not. Aspiration of fluid from the middle ears in the cases not improved by adenoidectomy restored normal hearing in most cases. As time went on the practice of tympanic aspiration at the time of adenoidectomy was adopted where serous otitis media was suspected. On the strength of encouraging results, accurate records were kept during 1957. This report concerns children who were seen in private practice in 1957 and in whom a diagnosis of serous otitis media was made. No cases of acute or chronic suppurative otitis media are included. The report may be considered inconclusive in that etiology is not definitely established. Inasmuch as all material here presented pertains solely to this group and for the sake of brevity, quotation from the literature is omitted.

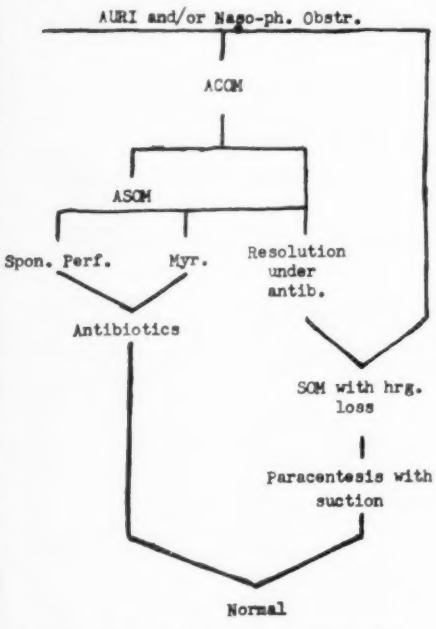
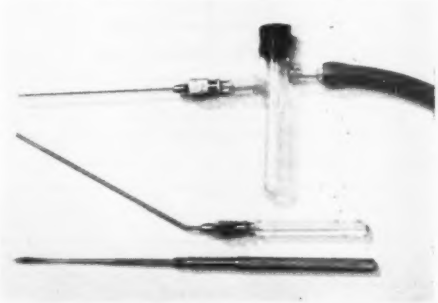


Fig. 1.—Likely origin or serous otitis media.

This condition collectively represents incomplete care of acute and subacute ear infections. The prevalence of the entity is now obvious. Most acute ear infections are treated with either adequate or inadequate antibiotic therapy, either of which relieves the distressing symptoms of pain and fever. It is only by evaluation a few weeks after the acute episode that serous otitis media can be diagnosed (Fig. 1 is self-explanatory).

Unilateral cases are easily overlooked, but the parent or school teacher usually recognizes hearing loss in the bilateral cases. It is most unwise to attribute this to inattention on the part of the child or to assume that he will outgrow his deafness. It is worth noting that every child studied in this series had received antibiotics for earache

Fig. 2.—Myringotome and aspirating equipment.



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Presented at combined meeting of the Philadelphia Laryngological Society and the Section on Otolaryngology of the Philadelphia College of Physicians on Feb. 4, 1958.

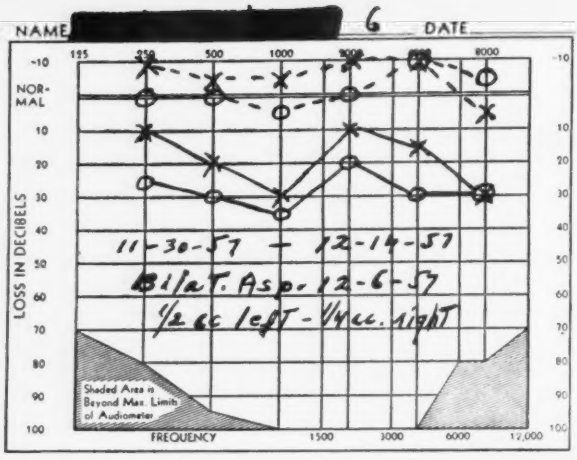
NAME [redacted] 6 DATE
NOR-MAL
LOSS IN DECIBELS
11-30-57 - 12-14-57
B1/aT. Asp. 12-6-57
1/2 cc left - 1/4cc. right
Shaded Area is Beyond Max. Limits of Audiometer
FREQUENCY
125 250 500 1000 2000 4000 8000
1500 3000 6000 12,000

Fig. 3.—Typical response to treatment.

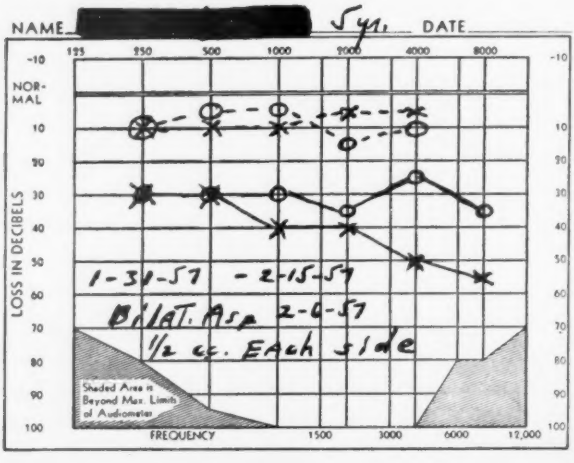

Fig. 4.—Typical response to treatment.

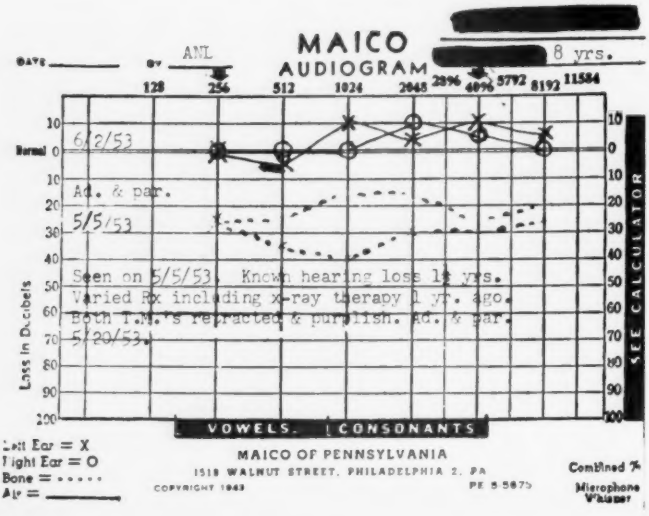

Fig. 5.—Typical response to treatment (1953).

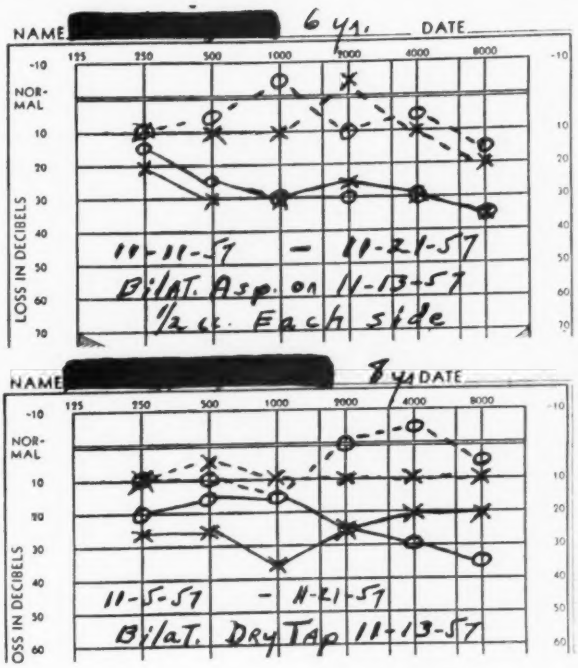

Fig. 6.—Response in brother and sister, one with dry tap.

within the year preceding diagnosis of serous otitis media.

Diagnosis is easily made, and the treatment appears effective. Etiology is questionable, and it is hoped that thoughts offered here will encourage additional investigation. Diagnosis is based first on the history of earaches and hearing loss, second on hearing evaluation, and third on the appearance of the eardrum. Loss of luster of the drum, however, is sufficient to arouse suspicion. It is by no means necessary that

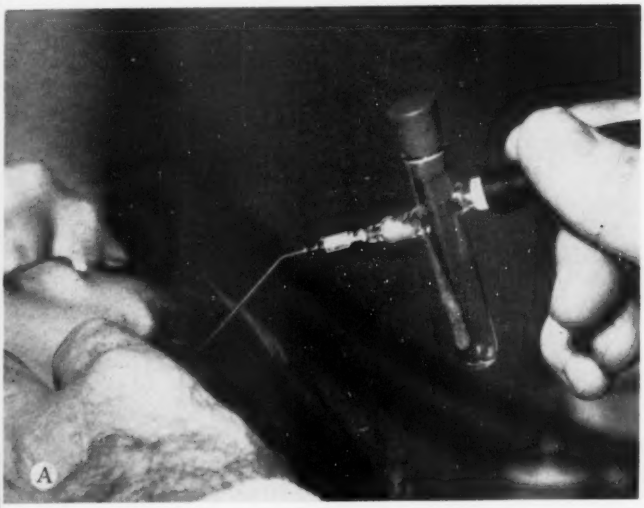

Fig. 7A.—Fluid aspirated from left ear.

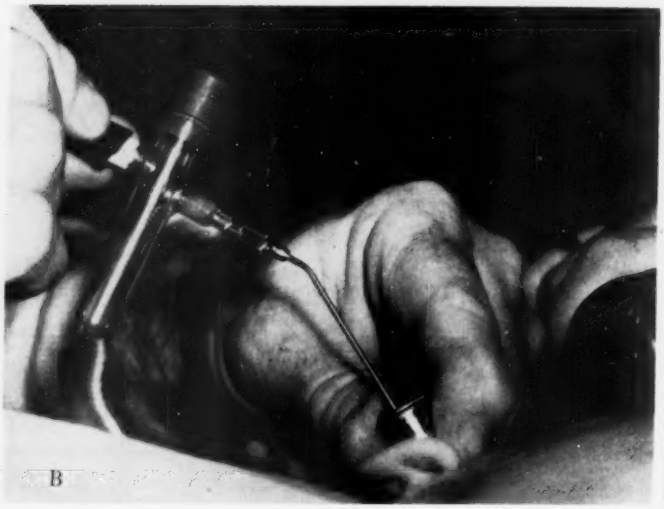

Fig. 7B.—Fluid aspirated from right ear.

B

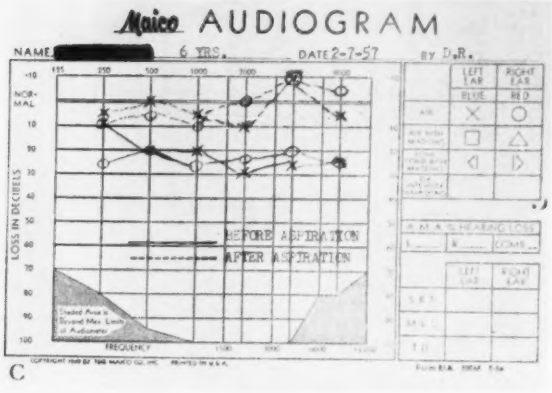

Fig. 7C.—Audiogram in case 7A and 7B.

C

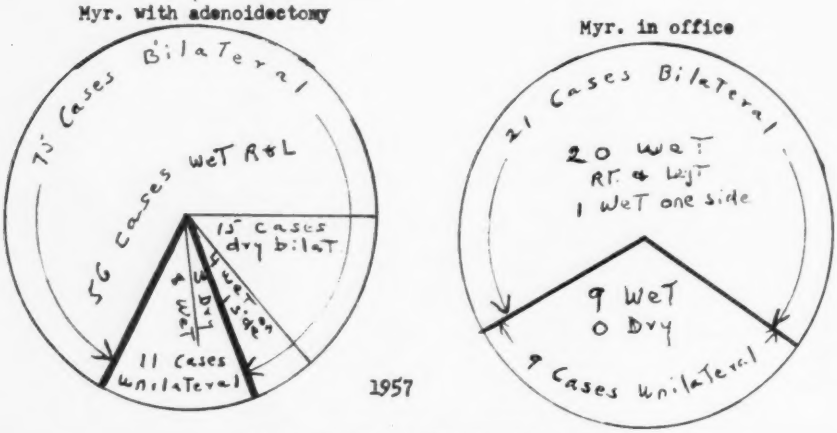

Fig. 8.—Findings in 212 aspirated ears.

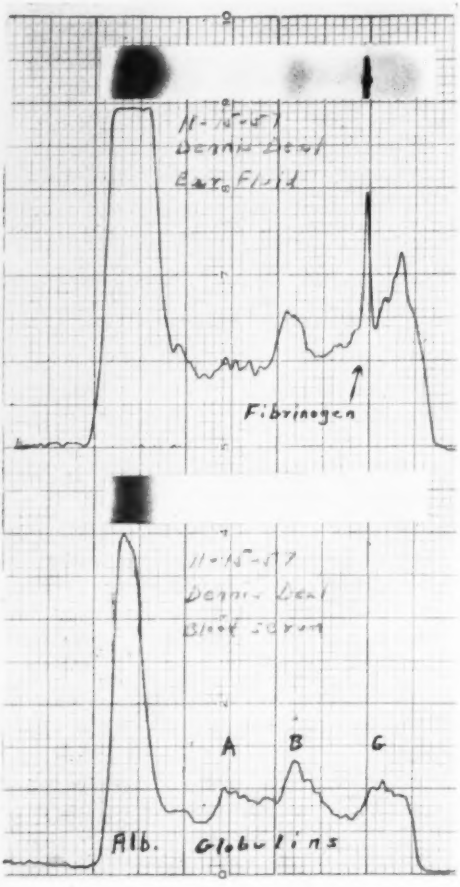

Fig. 9.--Comparison of middle-ear fluid and blood serum taken from the patient at the same time.

we see fluid level, bubbles, or color change, although such signs are corroborative. Audiogram shows a loss of 15 to 30 db. in all frequencies. Bone conduction was done on only a few children to avoid tiring the patient, but in those in whom it was determined the curve was considerably above the air-conduction curve.

The same treatment was employed in all cases. This consisted of paracentesis of the eardrum followed immediately by suction. Stab paracentesis with a myringotome was made in the anterior and in the posterior aspect of the inferior half of the drum. A 17-gauge suction cannula was held in contact with the two openings alternatively until no more material could be withdrawn (Fig. 2). A total of 116 children or 212 ears were treated in this manner; 86 cases were handled in conjunction with adenoidectomy, and 30 were performed under trichloroethylene (Trilene) analgesia as an office procedure. Of 212 ears, fluid was recovered in 174, and 38 proved to be dry.

The Table shows the manner in which the record is kept. Figures 3 and 4 show typical audiograms before and after the treatment described. Figure 5 is another typical case, in which treatment described was used in 1953 and is identical with the results obtained on others in 1957. Figure 6 shows audiograms before and after procedure in a brother and sister. Note that one was a dry tap. Figures 7A, 7B, and 7C

Myringotomy Record

Patient No.	Age, Yr.	Date	Procedure	Aspiration, Fluid	Diagnosis
1.	6	5/ 1/57	Left with A	½ cc. exud.	SOM
2.	7	5/ 2/57	Bilat.— Of.	½ cc. exud. bilat.	SOM
3.	4	5/ 3/57	Bilat. with A	½ cc. exud. bilat.	SOM
4.	4	5/ 7/57	Bilat. with A	Dry tap	SOM
5.	7½	5/ 8/57	Bilat. with A	½ cc. exud. bilat.	SOM
6.	5	5/ 8/57	Rt. with A	Dry tap	SOM
7.	6	5/10/57	Bilat. with A	½ cc. exud. bilat.	SOM
8.	9	5/ 9/57	Left—Of.	½ cc. pus	Acute
9.	2	5/11/57	Bilat.— Of.	½ cc. exud. bilat.	SOM 2d
10.	2½	5/11/57	Bilat.— Of.	½ cc. pus, bilat.	Acute
11.	7	5/13/57	Left— Of.	½ cc. pus	Acute
12.	5	5/16/57	Bilat.— Of.	½ cc. exud. bilat.	SOM
13.	4	5/18/57	Bilat. with A	½ cc. exud. bilat.	SOM
14.	7	5/18/57	Rt. with A	½ cc. exud.	SOM
15.	4	5/18/57	Bilat. with A	Dry tap	SOM
16.	4½	5/18/57	Bilat. with A	Dry tap	SOM
17.	8	5/21/57	Bilat. with A	½ cc. exud. bilat.	SOM

show fluid being aspirated from each middle ear and audiograms in the same case. Figure 8 is self-explanatory, showing a higher degree of accuracy in diagnosis among the aspirations done in the office. This is due to the greater temptation to open eardrums at the time of adenoidectomy.

Bacteriologic culture of fluid in the ears of 23 children showed no growth after three days in 16. Organisms found in the remaining seven cases were considered by the bacteriologist as unimportant or contaminants.

Figure 9 shows a comparison of middle-ear fluid and blood serum taken from the patient at the same time. Each is subjected to electrophoresis. Albumin and alpha, beta, and gamma globulin content is similar. Middle-ear fluid can seldom be aspirated free of blood; therefore, fibrinogen distorts the graph curve.

Examination of the fluid by the pathologist in 15 consecutive cases showed inflammatory exudate of high protein content.

Differential cell counts were made in nine specimens of ear fluid and comparison made with that of peripheral blood obtained from the patient at the same time. Much variation was found. In seven of these nine the leukocyte count was considerably higher in ear fluid than in peripheral blood. Differential count of ear fluid followed no consistent pattern. No eosinophils were found in any specimen.

Mastoid x-rays were taken in five consecutive cases in which fluid was recovered and in which there was no known previous ear discharge. In each of these the films showed moderate clouding of a normally developed mastoid.

Certain questions are anticipated, and the following statements may provide a partial answer:

1. Inasmuch as the treatment used seems effective, corticosteroids, antihistamines, or other drugs were not employed.

2. Recurrence can usually be avoided by removing lymphoid tissue from the nasopharynx at the time of tympanic aspiration. If aspiration is done and large adenoids left in place, hearing improvement is dramatic, but recurrence is almost certain. In 86 cases aspirated along with adenoidectomy only 2 had recurrences.

3. Only a very small proportion of children with serous otitis media show signs or symptoms of vasomotor rhinitis or asthma.

4. Myringotomy without aspiration is useless. The viscosity of the fluid is such that it will not flow out alone.

5. Bilateral aspiration was done in four consecutive adenoidectomies where middle-ear fluid was not suspected and all eight ears were found dry.

6. Treatment of serous otitis media by ear drops, nose drops, antibiotics, antihistamines, sulfonamides, physical therapy, and Eustachian inflation is without basis and is to be condemned.

7. The average age in this series was 6 years.

The following questions are yet to be answered:

1. Does the fluid penetrate the mastoid cells, and, if so, to what extent?
2. Does allergy play a part in etiology?
3. What happens to the fluid if not evacuated?
4. Is this a collagen disease?

Summary

A study is presented in which serous otitis media was diagnosed in 116 children.

Method of diagnosis with effective treatment is demonstrated.

Results of laboratory investigations are offered for speculation as to etiology.

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The Surgical Treatment of Chronic Suppurative Otitis Media

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Although chronic suppurative otitis media has been one of the oldest problems of otology, uncertainty still continues not only as to how to handle a given patient but also as to what constitutes the disorder. Diamant [1] considered it impossible to give a definition that would cover both the clinical and pathologic aspects of the condition known as "chronic suppurative otitis media." Attempts to define it as a perforation of the drum membrane with intermittent or chronic discharge resulted in such advice as that of Sir Charles Ballance,[2] who suggested that radical mastoidectomy with skin grafting should be done in any case of chronic suppurative otitis media for the safety of the patient. He based this advice on an analogy to chronic osteomyelitis of other bones and asked what surgeon would advise that a suppurating sinus tract be neglected. He was obviously lumping several distinct clinical and pathologic types of aural disease under one heading as the result of a faulty definition.

The analogy to chronic osteomyelitis of long bones is inexact. The mastoid process does not contain myelin, and the disease in the mastoid is usually acute osteitis, which only rarely becomes chronic. The analogy to chronic osteomyelitis of a long bone, therefore, holds good only in the rare instances of chronic disease of the petrous apex.

Ballance did not distinguish cases of perforations and simple mucoid discharge from cases of bone disease and cases in which cholesteatoma was the basis of the suppuration.

Wittmaack [3] by his theory of pneumatization provided further grounds for the tendency to operate on all types of chronically draining ears since he suggested that the small-celled and eburnated mastoids with their pathologic mucosa were doomed to continued suppuration.

Wittmaack's Theory of Pneumatization

Since its publication in 1918 Wittmaack's [3] theory of pneumatization has received considerable attention. He presented several hypotheses as follows: 1. The mucous membrane influences the development of the mastoid air-cell systems, and a normal mucosa results in a well-developed mastoid air-cell system. 2. During the birth process meconium or vernix caseosa may enter the middle ear and produce a pathologic type of mucous membrane, and infantile otitides may accomplish the same effect. 3. The pathologic types of mucosa cause total or partial inhibition or retardation of the process of pneumatization of the air cells which otherwise would have reached normal size, and these compact and small-celled mastoids are pathologic formations. 4. The pathologic types of mucosa govern the development of chronic suppurative otitis media.

These hypotheses were never well received by some authors, Cheatle,[4] Boies,[5] and Rüedi,[6] in particular, who pointed out the probable influence of developmental factors in the growth of the air-cell systems of the mastoid. Wittmaack's theory of pneumatization was completely demolished by Diamant in his monograph published in

Submitted for publication Jan. 31, 1958.

Read as Weiss Medical Lecture at the meeting of the Texas Society of Ophthalmology and Otolaryngology, Austin, Texas, Dec. 6-7, 1957.

Section of Otolaryngology and Rhinology, Mayo Clinic and Mayo Foundation. The Mayo Foundation, Rochester, Minn., is a part of the Graduate School of the University of Minnesota.

1952. Diamant found that infection of the mastoid was related to the size and development of the mastoid-cell systems, although he presented no hypothesis to account for it. However, the hypothesis of Wittmaack combined with the definitions of chronic suppurative otitis media suggested that in most instances operation was inevitable for this condition. As a result radical mastoidectomy was advised in cases in which the procedure added but little to the well-being of the patient, nor did it tend to ameliorate the mild symptoms present in many cases.

Lillie's Classification of Chronic Suppurative Otitis Media

In an attempt to solve this problem, Lillie [7] presented a classification of chronic suppurative otitis media in 1928, which, although modified from time to time, is still in use at the Mayo Clinic. He classified chronic suppurative otitis media into three types. The present version is as follows:

Type 1.—A central or marginal perforation of the tympanic membrane is present without evidence of bone necrosis or cholesteatoma formation. If discharge and infection are present, the mucosa of the middle ear, Eustachian tube, and mastoid antrum are involved. The discharge is not fetid.

Type 2.—Besides the perforation, necrosis of bone is present as evidenced by the odor of the discharge and the appearance of the ossicles and tympanic ring. This necrosis does not involve the mastoid process in a widespread manner. Polyps, springing from the necrotic ossicles, tympanic ring, or occasionally the promontory, may be present. Chronic suppurative otitis media of Type 1 or 2 does not justify mastoidectomy because it does not threaten the well-being of the patient, and problematic decrease in the discharge is the most benefit that can be hoped for from this operation.

Type 3.—The perforation of the tympanic membrane is usually marginal, and evidence of serious bone disease and usually of cholesteatoma is present. Roentgenograms of the mastoid may reveal not only ordinary sclerosis, but also definite evidence of a cholesteatoma in attic or mastoid antrum. In this type of chronic suppurative otitis media clinical judgment is needed. If signs of impending complications, such as pain in the head which awakens the patient from sleep, vertigo when the fistula test gives a positive result, recurring septic fever, and the like are noted, the need for surgical intervention is obvious. My colleagues and I have found, however, that even with the presence of cholesteatoma a number of these patients get along well under local medical treatment alone.

Perforations of the Drumhead of the Ear and Cholesteatomas

Diamant pointed out that, although benign perforation of the tympanic membrane is generally central, it may be marginal and located anteriorly and below the center. Classification of these perforations into marginal and central, therefore, is not of clinical significance. From the standpoint of clinical management, central and marginal perforations of the tympanic membrane when not leading into the epitympanum may be considered as being in the same category.

Marginal perforations in the drumhead lying superiorly and giving access to the epitympanum as well as perforations of the pars flaccida giving access to the same region should be considered separately, because in this type of chronic suppurative otitis media cholesteatoma may be present and surgical intervention may be imperative if dangerous complications are to be forestalled.

Wittmaack [3] stated the the superior marginal perforations are caused by tubal occlusion which produces a negative pressure in the epitympanic region. This negative pressure in turn produces a sucking in the pars flaccida. This tends to cause intrusion of an epithelial plug into the attic between the malleus and the incus. When this plug ruptures, inflammation destroys the mucosal lining, which is replaced by the ingrowing epithelium which forms the matrix of the

cholesteatoma in the attic. This may be spread laterally and involve the entire epitympanum, mastoid antrum, and mastoid cells, or may remain fairly well localized in the attic.

Diamant [1] maintained that marginal perforations do not occur at all as an effect of inflammatory processes. He stated that not a single case is on record in the available literature in which a marginal perforation of the "eardrum" (sic) has occurred as a sequel to acute otitis, nor has any case been reported in which a central perforation of the tympanic membrane has gradually developed into a superior marginal perforation during continued treatment for acute otitis. He emphasized that in marginal perforations an osseous destruction is found and must be found to render it possible clinically to establish the marginal character of the perforation.

These statements must depend on the fact that Diamant observed few cases of long-persisting acute suppurative mastoiditis in which sagging of the posterosuperior wall of the external auditory canal developed as a result of necrosis of the walls of the pneumatic cells of the mastoid located in this region. In my experience I have seen several fistulas develop in this region, usually lateral to the attachment of the pars flaccida. If these patients had not been operated on and chronic suppuration had been allowed to develop, I can see no reason why these superior fistulas leading into the epitympanum should not have persisted. In such cases epithelium from the external auditory canal might have grown in, causing an attic cholesteatoma. On the other hand, it might not, and the result would be a marginal attic fistula without cholesteatoma. According to Wittmaack [3] and Diamant,[1] there cannot be an attic fistula without an attic cholesteatoma, and yet such fistulas are observed not too infrequently. It is, however, true that the sucking in of Shrapnell's membrane has been observed in such cases, especially by Day.[8] Whether this is produced by tubal occlusion which usually results in serous otitis media or results from some other process is beside the point.

Diamant [1] demonstrated that islets of stratified squamous epithelium as well as a fully developed cholesteatoma may occur in the mucous membrane of the upper portion of the middle ear cleft without any simultaneous perforation of the drumhead or "chronic otitis." He stated that it is known that a cholesteatoma is sometimes a "genuine" tumor. He by no means claimed that a cholesteatoma is always a neoplasm. However, growing cholesteatomas in these regions can give rise to osseous destruction by a symptomless erosion. If this osseous destruction by erosion reaches the margin of the drumhead, it must be possible that the drumhead suffers destruction and a marginal perforation. The marginal perforation may be discovered by a secondary infection caused by bacteria invading the middle ear. A real otitis consequently develops, although it is not the cause of the perforation of the drumhead but is the effect of it. Diamant summed up by stating that the cause of perforations of the drumhead is unknown. He quoted Scheibe to the effect that a cholesteatoma is present in 91% of the marginal perforations of the eardrum and is not present in 9%. He stated, "Hence from many viewpoints it appears justifiable to regard the cholesteatoma as the primary factor in the development of marginal perforations."

Begley and co-workers,[9] in reviewing the problem of cholesteatoma, concluded that in all probability cholesteatoma can occur in the region of the temporal bone not only as a result of invagination of Shrapnell's membrane but also as a result of ingrowth of epithelium following an inflammatory perforation, by the activation of the anlage of squamous epithelium, by metaplasia of the cuboidal epithelium of the superior portion of the middle ear cavity, or as a primary tumor.

Whatever the origin of cholesteatoma, however, the decision as to what should be done about it when its presence is discovered seems more important than the origin.

Conservative Treatment.—With large attic perforations amounting at times to a radical mastoidectomy produced by the disease itself, with the cholesteatoma confined to the epitympanum, and a patient who is willing to return for frequent observation, it is my opinion that advancement of the lesion can be controlled and complications prevented for an indefinite period by conservative treatment alone. However, the only type of treatment which I have found effective is washing out the attic with a warmed alcoholic solution by means of a Hartmann syringe with a malleable silver cannula. Although this method of treatment is more than 50 years old, I am repeatedly surprised by the fact that few otologists seem familiar with it. This may be due to disinclination on the part of the modern otologist for the tedium of repeated treatments which are not especially spectacular. Yet I believe more treatment of this type should be done from the patients' standpoint, especially if the hearing is adequate for social and business purposes. In a favorable case, cholesteatomatous debris should cease to appear in the washings after a week, and the ear should become dry after two weeks and remain so. These patients should be inspected at intervals of six months, although the tendency to re-formation of cholesteatoma is variable. It has seemed to me at times to depend on the source of the cholesteatoma, that is, whether it originates from epithelial anlage or from metaplasia, or whether it has entered the middle ear, epitympanum, or antrum either by invagination of Shrapnell's membrane or by ingrowth of epithelium through a perforation.

If the attic perforation is too small for instrumentation or if the ear does not respond favorably to treatment, a Bondy type of radical mastoidectomy should be done.

Radical Mastoidectomy.—Before introduction of the sulfonamides and the antibiotics almost half of the patients with chronic suppurative otitis media seen at the Mayo Clinic had widespread destruction of the membrana tensa with marked disease or scarring of the mucosa of the middle ear and cholesteatoma involving the attic, antrum, and mastoid. Lately, however, this condition is rarely seen, and the majority of patients have superior perforations leading into the attic.

For the past 20 years my colleagues and I have been doing the Bondy modification of the radical mastoidectomy in this later type of case. In my earlier cases, influenced by the opinion of Nager that the matrix of the cholesteatoma would form an excellent epithelial lining for the radical cavity, I preserved the matrix as "the best thin epithelial lining." In some cases this seemed to be true, but in others the cholesteatomatous mass recurred rapidly, making necessary the frequent removal of cholesteatomatous debris from the cavity and causing occasional erosions of the horizontal semicircular canal or tegmen and cholesteatomatous cysts. It was and is impossible for me to forecast which matrix will become troublesome. In the last 10 to 15 years, therefore, it has been my practice to remove all vestiges of the cholesteatomatous matrix. In doing this I, like Juers,[10] have been impressed a number of times by the presence of finger-like projections of the cholesteatoma into the spaces around the semicircular canals. With the use of the fenestration type of flap, I can see no possible advantage to be gained by preservation of the matrix.

Experience with the use of Bondy's operation combined with the Lempert flap has been a happy one at the Mayo Clinic; the hearing usually is preserved and frequently is improved.

Operation for Chronic Mastoid Bone Disease of Children

One type of subacute or chronic mastoiditis, I believe, deserves special mention. It is a type seen rather infrequently, and, therefore, it is only in a large clinic that sufficient experience with it may be gained to come to a definite conclusion as to the manner in which it should be handled.

This type, which I prefer to characterize as chronic bone disease of children, seems to be becoming more frequent, possibly as a result of inadequate antibiotic therapy. These children present signs of acute mastoiditis with each upper respiratory infection. Roentgenograms of the mastoid indicate a small-celled mastoid with cloudiness and halisteresis of the cells on the involved side. At operation the mastoid cells are red and bloody, and the condition resembles the hemorrhagic mastoiditis of Beck and the "masked mastoiditis" of the days before introduction of the sulfonamides and antibiotics. However, if misled by this resemblance the surgeon decides to do a simple mastoidectomy, he must not be surprised to find his patient returning in a year or two with the entire mastoid region from the tegmen antri mastoideum to the sinal plate filled with a huge cholesteatoma even though there was nothing to suggest the presence of cholesteatoma at the original operation. In my opinion, patients with this type of chronic bone disease should have a Bondy type of radical mastoidectomy in every instance.

Operations for Nonotosclerotic Conduction Deafness

The surgical treatment for chronic suppurative otitis media heretofore considered has been for the purpose of preventing dangerous complications which might threaten the life or well-being of the patient. As a result of the surgical procedures for otosclerosis, the minds of otologists have turned toward the improvement of hearing in patients whose loss of hearing was owing to suppurative disease.

Early Efforts to Improve or Preserve Hearing in Cases of Chronic Suppurative Otitis Media.—Although attempts to preserve or improve the hearing by surgical intervention for chronic suppurative otitis media are by no means new, the introduction of the fenestration operation and the operation for improvement of hearing by mobilization of the stapes has resulted in new appreciation of the importance of loss of hearing as a handicap and has thus made all otologists more aware of the possibilities of modified or conservative radical mastoidectomy.

Heath,[11] in 1907, brought out his modified radical mastoidectomy for the conservation of hearing. He merely opened the antrum and cleaned it of granulations and debris and from the antrum attempted to curette the attic without removing the lateral wall of the epitympanum or disturbing the ossicles. He was never clear as to the indications for this procedure. He did state, however, that it could not be applied if a cholesteatoma was present in the attic. This, of course, greatly limited its usefulness.

Bondy,[12] in 1910, proposed that the lateral wall of the epitympanum should be removed, the bridge taken down, and the facial ridge lowered as close as possible to the attachment of the tympanic membrane. The ossicles were to be left undisturbed. He thought that the membrana tensa would seal and leave a closed tympanic cavity even if the pars flaccida was removed and the bodies of the malleus and incus were left protruding. In my experience this has not proved to be true, for a permanent opening usually remains between the ossicles. Mucopurulent drainage then develops with head colds, and the patient is dissatisfied because the ear does not become dry.

Stacke,[13] in 1911, stated that since 1898 he had been performing a radical operation somewhat similar to that described by Bondy. He found, however, that, even if the ossicular chain was absent or interrupted at the long process of the incus, improvement in hearing could be secured by attaching the membrana tensa to the body of the stapes. He also used Thiersch grafts to line the remainder of the cavity after a week or two and in this way assured himself of obtaining closure of the tympanic cavity.

In my experience, covering the bodies of the malleus and incus with a flap of skin from the meatus or a Thiersch graft tends to fix these structures, and the resulting lack of mobility tends to decrease the hearing.

Recently Introduced Restorative Measures for Suppurating Ears Together with Wullstein's Classification of Cases.—Zöllner,[14] Wullstein,[15] Pietrantoni and Bocca,[16] Maspetiol and associates,[17] and others have recently called attention to the possibility of improving the hearing when suppurative disease of the middle ear is still active or when injuries have proceeded further than simple perforation of the pars tensa.

Wullstein [15] has divided these cases into five types, but since these are essentially (1) the type in which the ossicular chain is intact, (2) the type in which there is a break in the ossicular chain, and (3) the type in which nothing remains but the footplate of the stapes, these three types are the ones considered herein. He also has a group in whom the round window niche or the oval window region or both are so bound down with scar tissue that mobility of the labyrinthine fluids is absent. He advised cleaning out the scar and covering the medial wall of the middle ear with mucosa from the cheek. Zöllner [14] stated that such mucosal grafts are invariably displaced in healing, and even Wullstein admitted that the results are disappointing. It is doubtful whether restorative operations are worthy of trial in such cases.

The great difference in the new classification of Wullstein and the old classification of Lillie is evident. The earlier is concerned with selecting the procedure which will best protect the well-being of the patient from the consequences of suppurative disease of the temporal bone. The later classification gives but passing concern to the pathologic condition except in so far as it influences hearing. I fear some of the hard-learned lessons of the past may be forgotten in the enthusiasm for restorative procedures.

In learning the technique of these newer procedures, the neophyte in otology finds himself on an equal footing with otologists of great experience. He may doubt, therefore, that the experience of the older man consists of more than multiple repetition of the same errors. However, those of us who have been fortunate enough to overlap both eras will find that the chronic running ear may still menace health and life and that enthusiasm for new and intriguing surgical techniques whose possibilities of securing hearing restoration have not been fully assessed should be tempered in the best interest of the patient.

However, it has been abundantly demonstrated that advances in knowledge of the physiology of hearing and the introduction of more powerful magnification have recently made it possible to improve hearing in cases in which destruction of the drumhead and transmissive mechanism of the middle ear has produced severe loss of hearing.

These physiologic principles have been clearly explained by Wever and Lawrence.[18] The restorative otologist is chiefly concerned with the necessity for a phase difference in the sound waves reaching the two windows, the fenestra ovalis and fenestra rotunda, the fact that there must be an elastic covering over each fenestra, and the fact that an air-containing cavity connected with a patent Eustachian tube must cover the round window. Successful restorative measures for the damages of suppuration and congenital malformation of the middle ear are largely based on these facts.

Closure of Perforations.—The closure of small central perforations of the pars tensa has been practiced for many years. The simplest and most effective method in my hands has been to cauterize the edges of the opening with 50% trichloracetic acid and then cover the perforation with a sheet of so-called Cargile membrane. The expected improvement can be foretold by the hearing gain produced by the application of the patch to the perforation. When the patch falls away, the process can be repeated.

Marginal perforations, however, cannot be closed in this way. House [19] was apparently the first to suggest closure of such perforations with a split-skin graft.

If the ear does not discharge and there is nothing to suggest cholesteatosis, after the surface epithelium is curetted from the

drum and the adjacent canal wall, by starting peripherally and working toward the edges of the perforation, the opening may be covered with a split-skin graft, and with light packing closure is usually secured.

Operations for Congenital Malformations.—This is a chapter in itself and is without the scope of this paper.

Technical Problems in Operations to Improve Hearing in Cases of Chronic Otitis Media.—The group of cases of otitis media Type 1 of Wullstein [15] (ossicular chain intact) includes those with attic perforations and attic cholesteatomas which have already been considered and also cases in which large central and marginal perforations are present but do not necessarily reach the attic.

Wullstein [15] recommended opening the antrum and inspecting the ossicles in such cases. However, if the ear is free of discharge and has good auditory reserve, I believe that simple closure of the perforation with a skin graft is the procedure of choice. In my experience, such ears almost never contain cholesteatomas, and inflammatory granulations, if any are present, can be expected to mature and clear up after closure of the perforation.

If suppuration is present, however, the Type 1 ear presents a different problem. Cultures taken of such ears almost uniformly show Pseudomonas and Proteus. These Gram-negative rods are extremely resistant to antibiotics, even to polymyxin. In preparation for tympanoplasty an attempt should be made to clean up the infection. In these ears I have obtained the best results by filling the canal and middle ear with 25% solution of silver protein (Argyrol) and then, using the finger as a plunger, forcing it through the Eustachian tube into the nasopharynx. This also serves as an assurance of a patent tube.

In approaching these ears I have used both the postauricular and endaural incisions and have found that both make the operative field equally accessible. The antrum is opened with the Hudson drill and Adson perforator, as this appears to combine safety with rapidity and at the same time gives an adequate exposure. The lateral wall of the epitympanum is taken away and the ossicles inspected. If the chain is intact and not bound down by adhesions, a portion of the bridge is preserved to protect the head of the malleus and incus from contact with the flap. In preparing a site for the flap the posterior wall of the canal should be well lowered and the tympanic bone flattened out.

In my earlier cases I used a full-thickness graft of postauricular skin, but I have found that a split-thickness skin graft serves equally well. The membranous meatus is cut through 1 or 2 mm. above the attachment of the drumhead, and the surface epithelium is removed with a chalazion curette as in a simple skin graft to the drumhead. The graft is inserted and packed in place similarly to the method used in the fenestration procedure.

Enlargement of the meatus is secured in endaural incisions by using my modification of the Lempert endaural incision.

For Type 2 ears (break in ossicular chain) the procedure varies little. If many scar tissue adhesions are present about the heads of the malleus and incus in the Type 1 ear, I believe that better results may be secured by converting the operation for the Type 1 ear into an operation for the Type 2 ear by removing the incus, cutting off the head of the malleus, and securing the columellar effect by pressing the graft firmly against the capitulum of the stapes, as originally suggested by Stacke, and recently termed "tympano-stapediopexy" by Juers.

If the long process of the incus is missing, the gap can be filled by mobilization of the ossicles, or if the gap is small, a piece of auricular cartilage may be inserted in the dehiscence. I do not think that the introduction of foreign bodies into the middle ear cavity will serve a useful purpose.

If the incus is entirely missing but the malleus and stapes are intact, it may be possible to dissect the manubrium mallei free from the drum membrane and to swing

the umbo into contact with the capitulum of the stapes.

In Type 3 ears when the footplate of the stapes alone remains and both windows are mobile, the problem is to protect the round window niche from the impact of sound waves, and to allow them to strike the stapedial footplate in a phase difference. At the same time an air-containing cavity connected with an open Eustachian tube must be secured.

In this instance, after the field is prepared, a thick graft must be placed as high up on the tympanic bone as possible with a fenestra over the site of the oval window. For this, a full-thickness skin graft should be used.

In my experience the use of absorbable gelatin sponge U. S. P. (Gelfoam) to preserve the middle ear cavity does not work well as Gelfoam is not absorbed and tends to become organized. If the mucosal covering on the medial wall of the middle ear cavity is intact, the raw surface of the flap will not adhere, and an air-filled cavity will be produced.

As far as packing is concerned, the same type of pack as that used in the fenestration operation for skin grafting of the radical cavity may be employed. Postoperatively the graft must be protected by adequate doses of antibiotics for from three weeks to a month. Inflation of the Eustachian tube should not be undertaken until at least two weeks after the operation so as to give the graft the best possible chance for a "take."

Illustrative Cases

The first two cases are those of a brother and sister with seemingly identical findings. I believe they illustrate several points about the possible genesis of attic fistulas, attic cholesteatomas, and the possible inheritance of mastoid types.

CASE 1.—This patient, a woman aged 32 years, presented herself at the clinic with the complaint of bilateral foul discharge from the ears. She stated that following bilateral otitis media in childhood she had experienced a continuous foul discharge from the right ear and that a year before admission a foul discharge had begun from the left ear also and had continued unabated until she presented herself for examination. She had a feeling of unsteadiness on bending forward, but no vertigo or dizziness on sudden motion of head or body. She never had exacerbations with fever, chills, pain in the head at night, or the like.

On examination the left drumhead was seen to be slightly retracted, and there was a large anterosuperior perforation into the attic from which a fetid discharge could be brought down with an incus hook. A similar condition was found on examination of the right ear, but there were granulations about the fistula. Culture of Escherichia coli was found on both sides. A positive fistula test could not be obtained on either side. The remainder of the ear, nose, and throat examination gave negative results, as did the general physical examination. Roentgenograms revealed infantile mastoids with sclerosis on both sides and the possibility of bilateral cholesteatomas.

A diagnosis of chronic bilateral suppurative otitis media was made, and radical mastoidectomy of the modified (Bondy) type was advised.

The mastoid processes were exposed by my modification of the Lempert endaural incision, and the antrums were opened with the Hudson drill. The right antrum and attic were filled with infected granulation tissue. No cholesteatoma was found, and the ossicles appeared to be normal and in perfect position. The ossicles were not disturbed, and a portion of the bridge was preserved over the ossicular heads to prevent their contact with the flap. A Lempert fenestration flap was turned over the facial ridge and bridge. The perforation was closed by pushing the edges together.

On the left side the antrum and attic were filled with a large cholesteatoma which had destroyed the incus and eroded the head of the malleus. The matrix of the cholesteatoma was removed completely, and a Lempert type of flap was turned down over the facial ridge and medial wall of the radical cavity. The fistula was handled in the same manner as on the right side. Attention was paid to securing contact between the flap and the capitulum of the stapes.

The cavities healed rapidly and without incident. Preoperative and postoperative audiograms showed that the hearing was maintained at about the preoperative level (Table 1).

CASE 2.—This patient, a man aged 36 years, stated that about once a year since childhood an acute exacerbation of bilateral otorrhea had developed and required rest in bed and antibiotic or sulfonamide therapy for two weeks. For several years he had noticed an increasing loss of hearing, especially on the right.

TABLE 1.—*Loss of Hearing in Case 1*

		Loss in Decibels at Various Frequencies							
		125	250	500	1,000	2,000	4,000	8,000	10,000
Preoperative									
A. C.*	Right	15	15	10	5	15	20	35	30
	Left	15	15	20	25	30	40	55	45
B. C.*	Right	—	10	5	10	5	10	—	—
	Left	—	15	10	15	10	15	—	—
Postoperative									
A. C.	Right	15	5	10	10	30	65	55	55
	Left	25	20	20	30	35	40	45	50
B. C.		Unchanged in both ears							

* A. C. = air conduction; B. C. = bone conduction in this and later tables.

On physical examination, the right tympanic membrane was partly concealed by a mass of polypoid granulation tissue which seemed to be coming from a defect in the wall of the attic and posterior canal. The tympanic membrane appeared to be relatively normal. On the left there was an attic defect with protruding cholesteatoma, the remainder of the drumhead appearing normal. A small quantity of fetid discharge was present on both sides. The remainder of the examination of the ears, nose, and throat did not contribute to the problem under consideration, and the results of the general physical examination, including roentgenograms of the chest and examination of the blood and urine, were normal. Roentgenograms of the mastoids revealed bony sclerosis on both sides with underdevelopment of the mastoid processes. The possibility of an attic cholesteatoma on both sides could not be excluded.

A diagnosis of chronic bilateral suppurative otitis media with cholesteatoma was made, and modified radical (Bondy) mastoidectomies were advised and carried out at separate sittings on the two sides. A modification of the Lempert endaural incision was used. On April 25, 1955, the left antrum was opened with a Hudson drill, and a cholesteatoma was encountered immediately. The lateral wall of the epitympanum was removed. The incus could not be identified, and the head of the malleus was much eaten away by the disease. The matrix of the cholesteatoma was entirely curetted away, and the remaining portion of the head of the malleus was bitten off. A Lempert flap was turned over the facial ridge and the medial wall of the radical cavity, and close contact of the flap to the capitulum of the stapes was secured.

On Sept. 26, 1955, the right ear was operated on in a similar manner. A large cholesteatoma was present which extended down to the tip and forward into the epitympanum, destroying the incus and the head of the malleus. The Lempert flap was turned down and fixed to the stapes in a manner similar to that used on the left side.

Healing was rapid on both sides, and the ears were completely epithelized and dry in three months. The postoperative audiograms showed a satisfactory gain in hearing on both sides (Table 2).

As stated previously, these two patients are brother and sister, and the fact that their mastoid processes appeared to be similar in the roentgenograms tends to support the hypothesis that the small-celled mastoid is a normal gene-propelled variant and is not the product of irritation either by meconium or bacterial invasion. This seems to controvert Wittmaack's hypothesis of inhibition of pneumatization. The findings in the woman of bilateral attic perforations, with cholesteatoma on one side and granulations only on the other, do not support Wittmaack's further hypothesis that retraction of the pars tensa is the normal cause of attic cholesteatoma, since the roentgenographic appearance was identical on the two sides, in spite of the fact that a cholesteatoma was found on one side and granulations on the other. It seems more reasonable to me to suppose that in this instance an attic fistula was produced by breakdown of the cells in the wall of the canal on suppuration with either a growing in of the epithelium from the external auditory canal or metaplasia of the epithelial lining of the attic on the side with cholesteatoma.

TABLE 2.—*Loss of Hearing in Case 2*

		Loss in Decibels at Various Frequencies							
		125	250	500	1,000	2,000	4,000	8,000	10,000
Preoperative									
A. C.	Right	10	5	15	20	25	45	50	40
	Left	15	10	15	25	45	50	55	55
B. C.	Right	—	0	0	5	10	5	—	—
	Left				Unchanged				
Postoperative									
A. C.	Right	10	5	15	20	20	25	30	20
	Left	10	10	15	15	20	25	35	30
B. C.		Unchanged in both ears							

TABLE 3.—*Loss of Hearing in Case 3*

		Loss in Decibels at Various Frequencies							
		125	250	500	1,000	2,000	4,000	8,000	10,000
Preoperative									
A. C.	Right	5	5	10	15	5	10	0	5
	Left	35	40	45	45	45	40	35	25
B. C.	Right	—	10	5	5	0	0	—	—
	Left	Normal							
Postoperative									
A. C.	Right	5	0	5	10	0	10	0	5
	Left	30	25	20	20	25	30	30	25
B. C.		Unchanged in both ears							

These two cases demonstrate that nothing is to be gained by preservation of the matrix of the cholesteatoma.

CASE 3. *Tympanoplasty for Type 1 Ear (Wullstein's Classification).*—This patient, a boy aged 10 years, stated that a year previously, without obvious cause, a copious quantity of foul pus began to drain from the left ear. No history of previous otitis could be obtained. The patient was given penicillin by mouth, and after six months the ear stopped discharging. It remained dry for a month or two, but the discharge started again.

On examination of the ears, nose, and throat the left external auditory canal was filled with thin foul pus, culture of which revealed Proteus vulgaris which was insensitive to tetracycline in concentration of more than 0μg. per milliliter. After cleaning, the tympanic membrane appeared granular and reddened. In the posterosuperior quadrant a polyp of granulation tissue covered a large portion of the tympanic membrane. The polyp seemed to be protruding through an attic perforation. Roentgenograms of the mastoid revealed marked sclerosis of the left mastoid, with some possible lack of development on the right. The remainder of the ear, nose, and throat examination and the general checkup were noncontributory.

Through a postauricular incision, the left mastoid was bared and the antrum was opened with a Hudson drill (Adson perforator). The antrum was found filled with purulent debris and a cholesteatoma. The lateral wall of the epitympanum was removed, and the epitympanum and ossicles were cleaned of cholesteatoma, including the matrix. The head of the malleus was partly eroded, but on elevation of the tympanic membrane the ossicular chain was found to be intact. The bridge was partly removed, but a sufficient amount was left to form a ridge pole. The drum and canal were freshened about the attic perforation with a chalazion curette; a full-thickness graft was placed over this area and the facial ridge and packed into place with absorbable gelatin sponge.

The postoperative period was without incident; the packing was sucked out in 10 days. The preoperative and postoperative audiograms indicated the improvement in hearing obtained (Table 3).

CASE 4. *Tympanoplasty for Type 2 Ear.*—This patient, a man aged 26 years, stated that he was in an explosion 17 years before registration, which left him with draining ears on both sides. He had had an operation of the right ear 15 years ago and since then had been unable to hear on that side.

Examination disclosed that the right tympanic membrane was completely destroyed, and a hook passed easily into the attic. The promontory was covered with velvety, red epithelium. A culture from the right ear revealed Pseudomonas aeruginosa sensitive to polymyxin at a concentration of 10 μg. per milliliter. On the left side there was a signet-ring remnant of the drum and ossicles, and the tremendous perforation was filled with scar membrane which was adherent to the promontory (a "natural" tympanoplasty). Roentgenograms revealed sclerosis of the right mastoid. The left mastoid was of the small-celled type and was normal. The audiogram revealed some loss of hearing (Table 4).

At operation the integument of the right canal was cut with the Lillie meatus knife as close to the attachment of the fibrocartilaginous ring as possible. Then, through a postauricular incision the mastoid was bared and the antrum opened with a Hudson drill. The lateral wall of the epitympanum was removed, and it was found that the incus, except for a remnant, was entirely destroyed. The posterior canal wall was flattened to the facial canal, the bridge was taken down, and the head of the malleus was cut off. The stapes was present.

TABLE 4.—*Loss of Hearing in Case 4*

		Loss in Decibels at Various Frequencies							
		125	250	500	1,000	2,000	4,000	8,000	10,000
Preoperative									
A. C.	Right	10	10	55	60	45	50	25	30
	Left	10	10	15	20	5	5	5	5
B. C.	Right	—	5	10	10	5	5	—	—
	Left	Sufficient masking to prevent lateralization to the right ear could not be obtained							
Postoperative									
A. C.	Right	35	30	25	30	25	45	35	30
	Left	Unchanged							
B. C.	Right	10	10	10	10	5	5	0	5
	Left	Unchanged							

but the crura sloped sharply caudally and were held in this position by scar-tissue bands. These were removed carefully, and the stapes was restored to its normal position. Then, the epithelium covering the fibrocartilaginous ring was curetted off. A small area over the promontory was bared of epithelium. The tympanic cavity was packed with small pieces of absorbable gelatin sponge soaked in hyaluronidase and penicillin solution. Then, a full-thickness graft was cut from the postauricular region and fitted into the cavity, care being taken to press it against the capitulum of the stapes as suggested by Stacke and by Juers to obtain a columella effect. The graft was packed in place with absorbable gelatin sponge, and a Korner type of plastic procedure was done on the membranous canal. The postauricular wound was closed.

The packing was sucked out on the 10th day and at two weeks the tube was cautiously inflated. The postoperative course was without incident (Table 4).

CASE 5. *Tympanoplasty for Type 3 Ear.*—A boy aged 5 years was brought to the clinic with the chief complaint of a chronically discharging ear on the right side. It was stated that the ear had discharged since infancy. Two years before admission, the patient had undergone a radical mastoidectomy reportedly of the Heath type. This operation had produced no change in symptoms.

On examination the left membrana tensa appeared normal. The right external auditory canal appeared normal, there being nothing to suggest a radical cavity. The drumhead was almost completely destroyed except for a "signet-ring" remnant with evidence of necrotic ossicles. The promontory was covered with thick velvety mucosa. A hook inserted into the attic brought down fetid cholesteatomatous debris. The general examinations yielded no other significant findings in the ear, nose, and throat. The audiogram showed great loss of hearing on the right. The bone conduction was normal in both ears.

A roentgenogram of the mastoids revealed postoperative changes and sclerosis of the right mastoid, the left mastoid appearing normal. Culture of the discharge from the right ear showed Escherichia coli and Micrococcus pyogenes. The Escherichia coli was sensitive to 20μg. of streptomycin per millimeter and 3μg. of a tetracycline per millimeter. The Micrococcus pyogenes was reported sensitive to penicillin more than 10 units per millimeter, to the tetracycline group, more than 6μg. per millimeter, and to erythromycin, 1μg. per millimeter.

Testing the hearing with an electrically driven acoustic probe showed marked improvement of hearing when the probe touched the region of the oval window.

Radical mastoidectomy with tympanoplasty was advised, but before surgical treatment was attempted it seemed advisable to free the middle ear and mastoid antrum from debris so far as possible. Attic irrigations with a saturated solution of boric acid in 50% alcohol were started, and after irrigation the middle ear cavity was filled with a 25% solution of silver protein (Argyrol), which was forced through the Eustachian tube into the nasopharynx by using the forefinger as a plunger in the external auditory canal.

The discharge from the external auditory canal disappeared in a week, but cholesteatomatous discharge continued to be brought down by attic irrigation. It was decided to proceed with the radical mastoidectomy and tympanoplasty.

Through an endaural incision the integument was elevated and the site of the previous mastoidectomy inspected. The antrum mastoideum contained infected granulations and cholesteatomatous debris. The bridge had not been removed, and most of the lateral wall of the epitympanum remained, especially anteriorly. Masses of cholesteatoma lay medial to the overhang of the lateral wall of the epitympanum. The antrum was enlarged, and the lateral wall of the epitympanum was removed; these revealed a cholesteatoma which surrounded the eroded bodies of the malleus and incus. The cholesteatoma and its matrix were carefully removed under magnification; the remnants of the malleus and the incus were taken out, and the bridge was removed. The region of the oval window was inspected under magnification. There was some granulation tissue in this region, and after meticulous removal the footplate of the stapes could be identified, but no trace of capitulum or crura could be found. The facial bridge was flattened down to the level of the descending portion of the Fallopian canal, and this flattening was carried down to include the tympanic bone, giving a broad flat base to give nutrition to the overlying skin graft. The epithelial lining was carefully removed from the remaining portion of the external auditory canal, but the fibrocartilaginous ring of attachment of the membrana tensa was carefully preserved. A skin graft, 25μ in thickness, was removed from the inside of the right upper arm with the electric dermatome. This is essentially a full-thickness graft. No scarification of the mucosa over the promontory was done even though it was thickened, as in my experience this is unnecessary as it will resume normal status after healing of the tympanoplasty. Neither was absorbable gelatin sponge placed in the eardrum. The flap was packed in place with paraffin mesh gauze. A small semilune of cartilage was removed from the conchal cartilage and the overlying integument turned under this edge and sewn posteriorly to give some enlargement of the external auditory meatus.

TABLE 5.—*Loss of Hearing in Case 5*

		Loss in Decibels at Various Frequencies						
		125	250	500	1,000	2,000	4,000	8,000
Preoperative								
A. C.	Right	50	55	60	60	50	50	45
	Left	0	0	0	0	0	0	0
Postoperative, 1 month								
A. C.	Right	20	20	30	35	30	35	25
	Left	Unchanged						

Adequate dosage of tetracycline was begun and continued for three weeks after operation. After a week the pack over the skin graft was removed, and the cavity was filled with an ointment containing prednisolone and polymyxin. After two weeks cautious inflation of the Eustachian tube was started and continued for a week. The patient was then dismissed on a maintenance dose of tetracycline. On his return in a month the radical cavity on the right appeared to be well healed, and the skin graft was in good condition. The audiogram taken a month after operation showed some improvement (Table 5).

While the improvement in hearing was not remarkable, it was significant and might prove useful. In addition the operation has produced a dry ear which should be safe from future complications.

Comment

These cases indicate that improvement of hearing in addition to healing of the suppurative process can be obtained in ears that might have been subjected previously to a classic radical mastoidectomy.

It is impossible for me to estimate the percentage of cases in which improvement in hearing can be obtained because of the relatively few that I have done. As experience and facility grow, undoubtedly better results are to be expected.

Summary and Conclusions

Attention to the importance of preservation of the hearing at operations for the chronic running ear has emphasized the importance of the closure of perforations, preservation of remnants of the tympanic membrane, and replacing of the destroyed drum with a skin graft when indicated.

These operations are based on the physiologic principles that an air-containing cavity communicating with an open Eustachian tube must be maintained to allow mobility to the round window membrane, and that the sound waves must not strike the windows in phase.

At the same time the older lessons in regard to the treatment of chronic suppurative otitis media must not be forgotten. The older classification of chronic suppurative otitis media has been supplemented but not replaced by the classification of Wullstein in regard to suitability for tympanoplasty.

This newer outlook on chronic suppurative otitis media has so influenced surgical technique that the so-called classic radical mastoidectomy is not indicated except in those instances in which hearing has been lost either through physiologic closure of the round window by scarring or because disease has invaded and destroyed the cochlear portion of the labyrinth.

Cases illustrating the tympanoplastic technique are given.

Mayo Clinic.

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Studies on Causative Factors of Mastoid Pneumatization

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Anatomical growth may be denoted to be the result of an interaction between hereditary and nonpathological environmental factors. In the general population, anatomical formations may be presumed to show a random size variation.

Pathological growth, in turn, may be denoted as the result of a disease entity interfering with the anatomical factors of growth. Pathological factors do not act alone in the determination of the final size. They must always combine with anatomical factors either to decrease or to increase anatomical size. This holds true even in agenesis actually caused by suppressing pathological factors.

Pathological influences will cause variations in size which may be expected to be distributed differently from the anatomical random variation. Pathologically caused size distributions may also differ, depending on the nature of the influencing pathology. Such differences may be due to many causes. For instance, the pathological influence may be exerted (*a*) in all individuals, or (*b*) in an appreciable number of individuals, or (*c*) in only a few individuals. It may operate (*d*) at the same rate in all individuals, or (*e*) at different rates in different individuals. It may be exerted (*f*) during development, or (*g*) after completed growth. It may cause (*h*) a halting of growth at any stage of development, or (*i*) a diminution of any size reached earlier.

The range of the *anatomical* size variation may be registered by studying large groups of individuals representative of the general population. The *pathological* plus and minus effect on the final size may be computed by comparing the variation in size caused by anatomical factors acting alone with that caused by anatomical and pathological factors in common. The selection factors for gathering the samples into representative groups must be meticulously watched in order to reach valid conclusions.

The first part of my [1] 1940 study aimed at determining the anatomical size variation of the mastoid air cell system. The selection factor used was the hospitalization for scarlet fever during the years 1932 to 1938, when hospitalization for this disease was still mandatory in Sweden. It was assumed that the individual susceptibility to scarlet fever was not related to the size of the mastoid air cell system. My large series was looked upon as a random sampling. It was expected to mirror the variation in size and the distribution of the various sizes within the general population.

My 1945 study with Dahlberg [2] aimed at determining what proportion of the total influence on the anatomical growth of the mastoid air cell system was caused by hereditary factors and nonpathological environmental factors respectively. The study was carried out on families and sets of uniovular and binovular twins. The selection factor was based on the fact that all Swedish children attend the unitary elementary school. Both children and parents were included in the study. Since it seemed justified to assume that the group represented a random sampling of the general population, the series was also used for checking the anatomical size variation found in my previous study.

The measurements obtained in the two studies gave basic information concerning (1) the size of each individual mastoid

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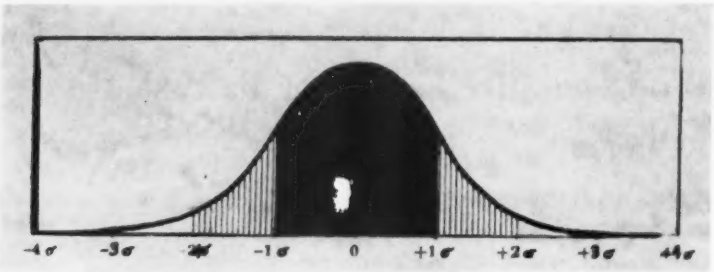


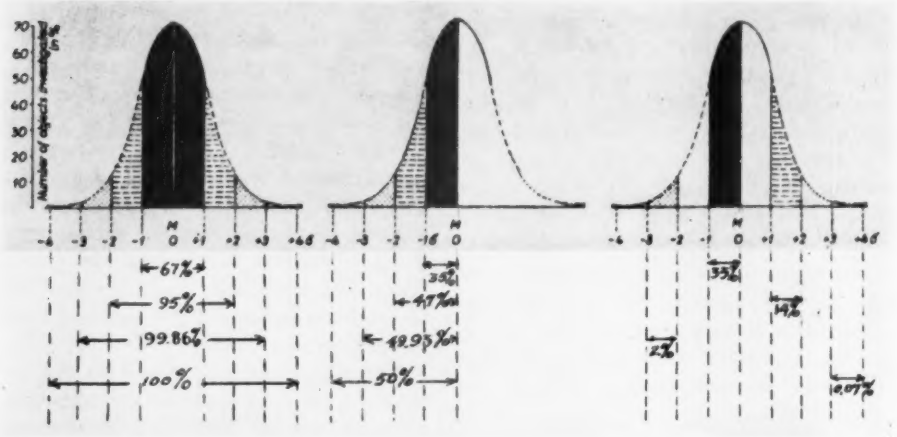
Fig. 1.—Curves of normal distribution. The mean is marked with 0. From the mean 1, 2, 3, and 4σ are plotted toward both sides.

air-cell system in the total sampling; (2) the range of the variation in size in the separate groups, and (3) the frequency distribution of the various sizes registered. In the separate groups the measurements allowed for the computation of (4) the mean size; (5) the standard deviation (σ), and (6) the standard errors. From the results of the studies conclusions were drawn which were assumed to be valid for the general population.

Samples collected according to a suitable selection factor in order to give a representative random sampling of the general population are commonly denoted by the term "normal material." The word "normal" in this designation has no medical meaning. The same holds true for the statistical terms "normal curve" and "normal variant." When speaking of the size of the mastoid air-cell system, and especially in studies aimed at determining the anatomical or pathological factors causing the development of the small mastoid air-cell systems, linking the terms "normal mastoid" and "healthy mastoid" may easily cause confusion. The use and misuse of the word "normal" in medical terminology has been discussed in a previous publication.[3]

The graphic distribution of the varying sizes registered in biological size studies is most commonly compared with the normal curve distribution (Fig. 1). On the ideal normal curve, the mean value is identical with the maximum frequency value, the so-called modulus. The mean also coincides with the median, i. e., an equal number of

Fig. 2.—Normal curve distribution. Variation in size or weight around the mean according to the computed value of σ; the mean is marked with 0.



samples falling on each side of the variation axis. The number of samples falling within each part of the variation axis on both sides of the mean, every part equaling the range of the standard deviation (σ), can be seen in percentages in Figure 2.

According to my introductory definitions, hereditary and nonpathological environmental factors by their interaction cause the anatomical growth and the anatomical final size variation always found in the general population. It is a common experience that any biological interaction of this kind may result in a random size variation after completed growth. Provided that this is accepted, the varying sizes of the mastoid air-cell system in a sufficiently large series representative of the general population may, as a general rule, be expected to be distributed in close conformity to the ideal normal curve.

The degree of conformity between the frequency distribution of the various sizes actually found in the study and the ideal normal curve can be computed. It often forms the basis for conclusions as to the factors influencing growth. However, to judge whether anatomical factors alone, or anatomical and pathological factors in common have been influencing the growth involves a very complicated process, and the final conclusion cannot be based solely on the conformity or nonconformity to the normal curve distribution.

Conformity to a normal curve is in itself no proof that anatomical factors alone have been influencing growth. Theoretically, conformity of any two curves may be coincidental. But, if a conformity to the normal curve is found in two separate studies carried on with two different samples of normal material, as is the case in my studies of 1940 and 1945, then this agreement may be claimed to support the conclusion that only anatomical factors have been influencing growth. Repeated conformity to the normal curve may hardly be maintained to represent a coincidental finding.

On the other hand, the nonconformity to a normal curve distribution is no proof that anatomical factors have *not* alone been determining growth and final size. First, an absolute conformity can hardly ever be expected. Among the disturbing factors may be mentioned the necessity of restricting the number of samples collected for

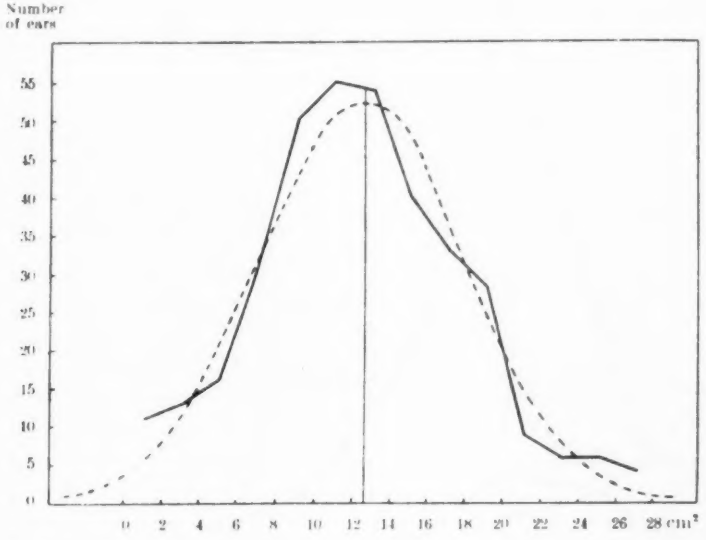


Fig. 3.—Distribution of the size of the air cell system (356 normal ears). The corresponding normal curve is also given (dotted line).

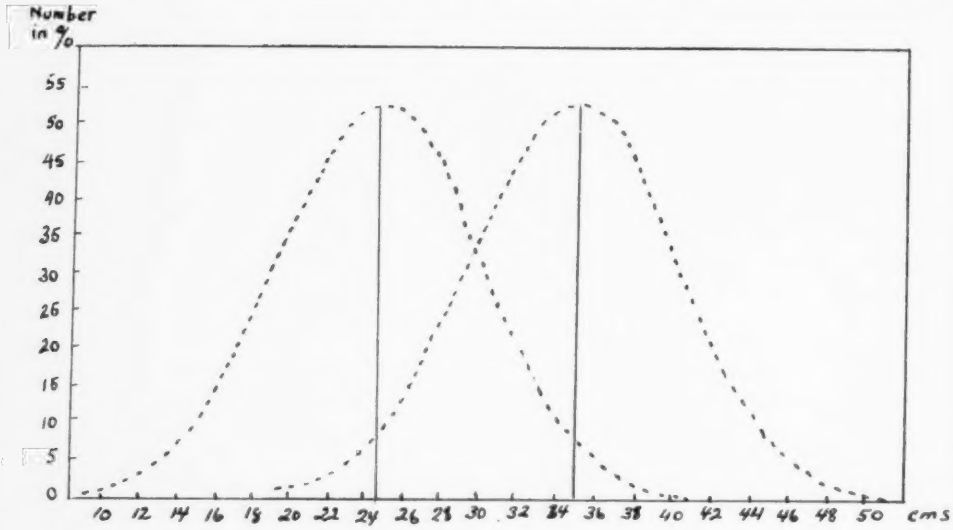


Figure 4

investigation while still keeping it a representative group, as well as unavoidable inaccuracies in the registration method itself. Secondly, an obvious nonconformity to the normal curve, as obtained in my chronic ear disease series in 1940 (Fig. 5), cannot per se justify the conclusion that anatomical factors alone have *not* been deciding growth and final size of the mastoid air-cell systems in the chronic ear disease group.

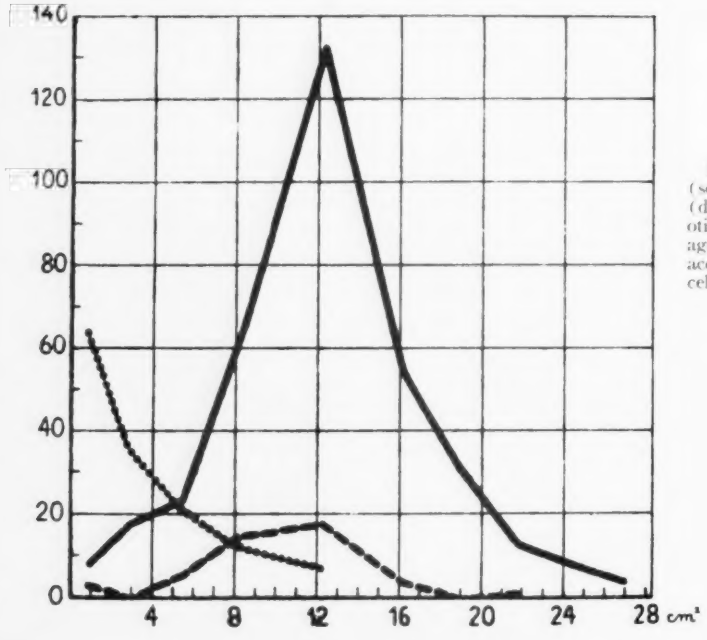


Fig. 5.—Normal material (solid curve), chronic otitis (dotted curve), and acute otitis (broken curve) at the ages of 10-30 years, grouped according to the size of the cellular system.

The distribution curves resulting from studies of the variation in size of the mastoid air-cell system have been shown to fall into three different types: (1) a close conformity to the normal curve (Fig. 3), (2) a shift to the left, still in close conformity to the normal curve (Fig. 4), and (3) a "skewness" not at all conforming to the normal curve (Fig. 5).

The close conformity to the normal curve, as well as the two deviating curves, may all represent anatomical factors, exclusively, which have influenced growth and final size. The normal curve is to be expected according to definitions and the selection of a random series representative of the general population; the shift to the left may represent an incomplete stage of growth or a sex difference, and the "skew" curve may be the effect of the selection factor inducing an overrepresentation of certain sizes in the study.

On the other hand, all three curves may also be found when pathological factors have actually interfered with the anatomical growth. The normal curve distribution may be a coincidental finding; the shift to the left may occur when all sizes in the study have been pathologically influenced to about the same extent, and the "skew" curve may occur when an appreciable number have been pathologically altered.

Whatever significance the above difference between the two *deviating* curves may be said to represent, the presence of a deviating curve does not in itself allow the conclusion that a pathological factor, and still less that a disease entity has actually interfered with the anatomical growth and altered the final size. The presumed existence of a pathological influence must be confirmed by other elements in the study; its presence can then be supported by the finding of a deviating curve distribution.

The clinical presence of chronic *mastoiditis* has been claimed as the cause of a gradual shrinking in size of the mastoid air-cell system. The occurrence of manifest chronic *mastoiditis* is rare in the ear clinic[4] and doubtlessly far below 1% in the general population. Consequently, in a study of 300 ears in a group representative of the general population three ears at most can be expected to be pathologically diminished in size in this way. Chronic *mastoiditis*, therefore, cannot be claimed to represent a significant cause for the development of small mastoid air-cell systems in the general population where 50% of all persons show a size falling short of the mean size. Even the occurrence of three cases does not cause any significant deviation from the normal curve distribution which may be expected to be obtained from the remaining 297 ears. Thus, the presence of chronic *mastoiditis* is a poor explanation of a deviating distribution curve when such a curve is obtained in a study not having chronic *mastoiditis* as the selection factor for the sampling. We may conclude from this that the significance of the conformity or nonconformity to the normal curve distribution in studies of the size variation of the mastoid air-cell system must always be judged in close relationship to the selection factor used for gathering the representative group samples for the study. It must be remembered that the distribution curves obtained in the studies have a somewhat different significance when looked upon from the aspect of the curve itself and from the aspect of the selection factor used.

A. From the aspect of the distribution curve itself
   1. The close conformity to the normal curve indicates that anatomical factors, exclusively, have determined growth and final size, provided that the conformity is not merely coincidental.
   2. The shift to the left (or right) indicates either (*a*) that anatomical factors, exclusively, have decided the growth and final size, the deviation of the curve being an effect of uncompleted growth at early ages or of differences between the sexes, or (*b*) that pathological factors have interfered with growth and final size, provided that all sizes have been influenced to about the same extent.
   3. The "skewness" of the curve indicates that the selection factor used has induced an overrepresentation of certain sizes in the

group samples collected for the study. This means either (*a*) that anatomical factors exclusively have decided the growth and final size, or (*b*) that pathological factors have interfered with growth and final size, provided that all sizes have *not* been influenced to about the same extent.

Conclusion: Under no circumstances is it possible to judge from the aspect of the distribution curve *which* factors, anatomical or pathological, represent the actual influence on growth and final size of the mastoid air-cell system.

B. From the aspect of the selection factor itself

1. A large series of samples representative of the general population, the normal material, may be expected to present a close conformity to the normal curve distribution. This is a consequence of the definition of anatomical growth which permits the assumption of a random size variation.
2. A large series of samples selected by any chosen selection factor in order to give a group representative of a disease entity may be expected to present a deviation from the normal curve distribution, provided that the selection factor induces an overrepresentation of certain sizes in the total range of variation. The deviation may be caused either (*a*) directly by using a selection factor which itself singles out a certain size, for instance, the collection of radiograms of acellular mastoids, or (*b*) indirectly by using a selection factor which itself does not single out certain, say, small sizes, but nevertheless has that effect, for instance, the collection of ears with tympanic membrane perforations.

Conclusion: Under no circumstances is it possible to judge from the aspect of the selection factor *which* factors, anatomical or pathological, represent the actual influence on growth and final size of the mastoid air-cell system.

It has already been stressed that the selection factor must be meticulously watched in order that valid conclusions may be reached. The use of unsuitable selection factors may invalidate the final data by (1) not giving the selection aimed at, and (2) giving a selection not aimed at. We may take a few examples from mastoid air-cell system studies illustrating expected and unexpected effects of the choice of the selection factor.

I. Selection factor: tympanic membrane perforation. This selection factor must give a marked overrepresentation of small air-cell systems, since it has been shown that no tympanic membrane perforation does occur in ears with an air-cell system size exceeding the mean size of the air-cell systems in the general population.

II. Selection factor: random sampling of patients from an ear, nose, and throat department clinic. This selection factor must give some overrepresentation of small air-cell systems for two reasons:

(*a*) It will induce an overrepresentation of patients with tympanic membrane perforations as compared with the general population (the effect noted under I.) and

(*b*) it will induce a further overrepresentation of small air-cell systems, since it has been shown that otitis infections do not occur in the largest air-cell systems in the normal material (i. e., in a sampling representative of the general population). Consequently individuals with very large air-cell systems will be less often seen in an ear, nose, and throat department clinic.

III. Selection factor: "healthy" individuals selected *not* to show signs of previous or present otitis infections. This selection factor must give some underrepresentation of small air-cell systems, since, as compared with the general population, i. e., the normal material, some of the small air-cell systems must be excluded.

IV. Selection factor: poverty. This selection factor must give some overrepresentation of small air-cell systems, since poverty represents various disabilities preventing success in the community. Among the many causes of disability a prominent one is hardness of hearing. Hearing loss is often caused by discharging diseases of the middle ear. The sampling of middle ear discharging diseases induces an overrepresentation of small air-cell systems, since "chronic otitis" is found only in ears with a small air-cell system (see under I and II).

V. Selection factor: slum school children. This selection factor must give some overrepresentation of small air-cell systems, since hereditary factors influence the determination of the size of the air-cell system (Dahlberg and I have shown that hereditary factors are the preponderant influences) and since poverty as a selection factor induces an overrepresentation of small air-cell systems in the parents of slum school children (see under IV).

VI. Selection factor: telephone subscribers. This selection factor must give some underrepresentation of small air-cell systems, since telephones are uncommon in the slums and common in the wealthy classes. The result of using this selection factor will consequently be the opposite of that of poverty and slum school children (see under IV and V).

The drastic example of telephone subscribers as a selection factor for studies on size of the mastoid air-cell system has in-

tentionally been added to the series of varying selection factors actually used. It is intended to illustrate the fact that it is impossible to attribute etiological properties to any of the selection factors used even if they really can be shown to cause an overrepresentation of small air-cell systems. Whatever the result of the selection factor may be, an overrepresentation or an underrepresentation of certain sizes, the result does not substantiate the conclusion that the selection factor itself influences the growth, i. e., interferes with the anatomical growth. Even when the selection factor definitely represents pathological factors or is closely associated with such factors, the resulting distribution curve of the various sizes of the mastoid air-cell systems in the study does not substantiate the conclusion that pathological factors have interfered with the anatomical growth or final size. Still less does it substantiate the conclusion that a certain disease entity, for instance, otitis infection, has determined or even influenced the growth of the mastoid air-cell system. The effect of pathological factors on the anatomical growth of the mastoid air-cell system can under no circumstances be shown in this way.

Nevertheless, a meticulous watching of the selection factor is important. The importance can be demonstrated in the following example:

The clinical diagnosis of "chronic otitis" encloses a nonhomogeneous group. Some of the problems concerned with this group include the question of the etiology of the central and the marginal tympanic membrane perforation and of the nature of the cholesteatoma. The selection factor for the series of chronic discharging ears in my study in 1940 was based on the presence of tympanic membrane perforations. A further classification was made according to the site of the perforation, central or marginal. By using this division I expected to find out whether or not these two groups, each being clinically homogeneous in its pathology, differ significantly in their relationship to the size of the mastoid air-cell system. A significant difference was actually found to be present. The marginal perforation did not occur when the size of the air-cell system exceeded 10 sq. cm. The central perforation did not occur when the size of the air-cell system exceeded 15 sq. cm.

The coexistence frequently shown to exist between middle ear discharge and small mastoid air-cell systems, including acellular mastoids, has raised two fundamental questions concerning the causative factors of mastoid pneumatization: 1. Is there a cause-and-effect relationship between otitis and the size of the mastoid air-cell system? 2. If present, which is the cause and which the effect?

The classical phrase "post or propter" indicates that studies designed to solve problems of medical cause-and-effect relationships are difficult to plan and execute. It also indicates that final proofs can never really be ascertained. Therefore, even findings and conclusions of rather comprehensive investigations are usually open to objections. The studies on otitis and mastoid pneumatization may serve as an example.

My study in 1940 was the first to establish knowledge of the anatomical variation in size of the mastoid air-cell system as well as the distribution of the various sizes in the general population. The sizes were found to vary in a continuous sequence from 0 to about 30 sq. cm. as measured with a planimeter on the roentgenogram in lateral projection. The mean size was about 15 sq. cm. The plotting of the measurements showed their distribution to conform close to a normal (probability) curve (Fig. 3). It could be verified (by my study in 1945) that the conformity was not coincidental. In clinical examination only a few ears in the large series showed any signs of previous or present otitis infections.

The conclusion was drawn that all sizes of the mastoid air-cell system may be normal variants and that anatomical factors

alone may be able to produce all sizes of the air-cell system actually found.

Two important facets of the mastoid pneumatization problem could not be revealed by the study: first, the exact nature of the environmental factors influencing growth and final size; second, the possibility of otitis playing a role in the determination of the final size.

The knowledge obtained from the normal material together with that obtained from the various groups of otitis material made it possible, however, to compare the variation in size of the mastoid air-cell system in the general population with the variation found in individuals presenting central and marginal perforations.

The range of the variation in size in each of the pathological groups was found to fall completely within the size range representative of the general population. Furthermore, it was found that all the pathological ears had air-cell systems smaller than the mean size computed for the general population. As has already been mentioned, the upper borders for ears with marginal and central perforations were 10 and 15 sq. cm. respectively.

The two groups of pathological middle ears thus showed a marked overrepresentation of small air-cell systems as compared with the group representative of the general population. It could also be shown, by computation of relative risk figures [5] that the volume of this overrepresentation increased rapidly with decreasing sizes of the air-cell system.

As regards the problem of the cause-and-effect relationship between otitis and size of the mastoid pneumatization, my 1940 study showed that the coexistence frequently found in an ear department clinic between tympanic membrane perforation and the small mastoid air-cell system varies significantly with different selection factors used for the registration:

(1) If one collects 100 ears clinically showing tympanic membrane perforations, one will find that the air-cell systems in all of them have a size below the mean size of the general population. They will, of course, also show signs of ear disease, as, for instance, impaired hearing added to the existent tympanic membrane perforation.

(2) If one collects 100 ear roentgenograms from a set of roentgenograms taken for other reasons than existing ear disease, selecting only those presenting a size of the air-cell system smaller than the mean size computed for the general population, one will as a rule find, at the clinical examination of the corresponding individuals, no ears showing tympanic membrane perforations. Furthermore, practically all of them will show no signs of previous or present otitis, or any evidence of impaired hearing.

Thus, the coexistence between the small mastoid air-cell system and the tympanic membrane perforation is not obligatory. If the selection factor used is the presence of a tympanic membrane perforation, it will appear, however, as if this coexistence were inevitable.

At first sight the knowledge obtained in my study looks suitable for *excluding* otitis as playing a role in the determination of the final size of the air-cell system. This, however, is not without objections, some of which may be justified. First of all, conclusions can be valid only for the selection factor chosen, which in this case is the tympanic membrane perforation. Therefore, otitis processes not causing a tympanic membrane perforation cannot by my study be excluded as causing an influence on the variation in size of the air-cell system.

Theoretically it is still possible, therefore, to claim that *manifest* otitis infections have influenced the size. Even the fact that the general population shows a range of variation comprising all sizes and that the variation in the general population may be expected to represent an effect of anatomical factors exclusively, does not exclude the possibility that some of them have been coinfluenced by pathological factors. This has already been stressed in my 1952 publication [5] and is also discussed here under the possible effect of chronic mastoiditis.

As a matter of fact, it may even be claimed that a shift to the left exists in the normal curve distribution obtained from my normal material. This would be the result of a combined influence of anatomical factors and pathological factors, the latter exerting their influence to about the same extent in all air-cell systems.

Moreover, it has in fact repeatedly been claimed in the otological literature that *aseptic* otitis processes have a retarding effect on the growth of the mastoid air-cell system, and also that such aseptic processes are universally present during fetal life. Some authors have even claimed that otitis processes, septic or aspetic, may pass clinically *unobserved*, leaving as the only sign of their previous existence a halted growth of the mastoid air-cell system. In this way, the very presence of a small air-cell system is claimed as proof that pathological factors have interfered with growth. Asymmetry, according to this theory, is always pathological. In asymmetry, moreover, always the *smaller* air-cell system is claimed to have resulted from the presumed pathological influence on growth and final size.

The above claims have all been made in the otological literature and sometimes in connection with objections to the conclusions drawn from my 1940 and 1945 studies. It is obvious that all objections to conclusions must be based either on disagreement concerning selection factors, methods of investigation, or the conclusions themselves. It is, however, just as obvious that objections to my "anatomical variant theory" do not constitute scientific support for a "pathological" theory. Any theory claiming a pathological influence on growth of the small mastoid air-cell systems itself requires confirmatory studies to prove its veracity.

It may be stressed in this connection that one important finding in my 1940 study seems to stand unopposed. No otitis process, be it clinically observed or unobserved, be it septic or aseptic, is claimed to cause a permanent tympanic membrane perforation in ears in which the air-cell system exceeds the mean size computed for the general population.

My 1945 study with Dahlberg comprising representative family groups as well as sets of uniovular and binovular twins allowed of calculations indicating that all sizes of the air-cell system are preponderantly decided by hereditary factors. Environmental factors were also shown to exert an influence, though to a slighter degree. Some support was obtained for the conclusion that the environmental factors exerted their influence mainly prenatally.

No investigator has claimed that hereditary factors have no influence on the determination of the final size of the mastoid air-cell system, i. e., that nonpathological or pathological environmental factors represent the sole influence on growth of the mastoid air-cell system.

The objection has been raised, however, that the calculations performed in my 1945 study with Dahlberg do not allow of the conclusion that the environmental factors exert their influence mainly prenatally.* But even if this objection were justified, i. e., even if the environmental were not *shown* to represent a prenatal influence, this in itself does not prove that they do *not* act prenatally, or that they do act exclusively postnatally. Still less does it prove that the influencing postnatal environmental factors are pathological, or that they represent otitis infections, septic or aseptic, observed or unobserved.

It goes without saying that the exact nature of the prenatal or postnatal environmental factors could not be disclosed by Dahlberg's and my study on heredity. It

* It has been maintained that since our calculations were based on the mean size of both of the individual's ears, they did not show the real difference between the uniovular and binovular twins. As a matter of fact, we compared the means of the means in our study. Since the two twin groups in the study were numerically fairly large, it seemed possible to conclude that the difference shown to exist was not coincidental but was caused by a real difference in asymmetry between the groups of twins.

must, however, be quite obvious that the objection that we have not defined their exact nature is completely irrelevant. Any deductions concerning these factors lacking support of scientific investigations would of course be mere guesswork. The proclamation that they are pathological and represent otitis infections must likewise represent mere guesswork if not supported by thorough investigations.

A study could be planned to reveal the absolute percentages of clinically manifest otitis of various forms occurring during the whole lifetime in all individuals of the general population as related to the size of their mastoid air-cell systems. But even this study, if at all possible to perform, would still not exclude objections claiming that the "normal variant theory" is wrong because unobserved, and aseptic otitis processes have not been excluded as decisive in influencing the final size of the small mastoid air-cell system. The burden of proof must always rest with the author proclaiming as his working hypothesis that pathological factors are responsible for the optional growth of the small mastoid air-cell system.

When one looks for studies claimed to support the "pathological theory" it seems at the present time to be commonly accepted that Wittmaack's (1918) pneumatization theory does not support the premise that the small mastoid air-cell system is pathological. Disregarding the question as to whether studies under the microscope are apt to disclose causative factors of macroscopical formations and their macroscopical variations in size, Wittmaack himself repeatedly and emphatically denies that he has drawn conclusions concerning the *size* of the mastoid air-cell system. He stresses the fact that his conclusions are valid for the *irregularity* of the air-cell system, and that this irregularity may be found in all sizes, from the very smallest to the very largest.

A different approach has recently been tried in an effort to reveal the influence of environmental factors on the mastoid pneumatization. Experimental studies on the pneumatization process have been performed in animals. In this way it was hoped that, by varying the environmental factors experimentally, the possibility or impossibility of disturbing the ordinary pneumatization process could be disclosed. It goes without saying that conclusions drawn *ex analogiam* from animal experiments are limited in many ways and must be carefully watched, especially as regards the mastoid air-cell system in man. The possibility of disturbing growth experimentally, moreover, does not prove that such disturbances occur in the actual anatomical or pathological development.

In the experiments the function of the tuba auditiva in dogs and guinea pigs has been suppressed; the bulla ethmoidalis in guinea pigs has been infected, and the foramen pneumaticum in the chicken humerus has been destroyed or infected. It seems from the experiments that the stopping of the pneumatization in the chicken humerus is difficult even by the induction of severe lesions. On the other hand, interferences with vital stages of the pneumatization process equivalent to cessations were effective and caused a complete inhibition of pneumatization. It may be stated that where the induced infections succeed in influencing the pneumatization process, either by a delaying or by a real diminution of the final size, all experiments showed that additional local signs arose together with the effect caused by the experimental process. In drawing conclusions from the experiments on the mastoid air-cell system in man, above all this important fact seems to have been completely disregarded. About 50% of all persons have air-cell systems smaller than the mean size of those of the general population. These persons only rarely show any signs whatsoever of ear pathology, functionally, otoscopically, or radiologically. They also only rarely give histories of previous otitis infections. This last statement points out that the possibility of disturbing the pneumatization process in animals by strong environmental interferences cannot logically be applied to the

ordinary pneumatization development of the mastoid air-cell system in man.

In 1939 I suggested that the chicken femur and tibia, obviously showing varying rates of pneumatization, offer apt material in which to study the causative factors of the pneumatization process. I emphatically reiterate this suggestion. However, the study of the ordinary pneumatization process would be more conclusive than the study of an experimentally disturbed (or induced) pneumatization process. On poultry farms it could be easily arranged to cross breed hens with pneumatized and nonpneumatized femurs and tibias in order to obtain material for a study of the resulting rate of pneumatization in successive generations. This suggestion should provide a fruitful and rewarding study for some young investigator interested in pneumatization and otitis problems.

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Excessive Forward Projection of the Nasal Tip in Rhinoplasty

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The technique for lowering a nasal tip which projects too far forward is difficult from a technical and an artistic standpoint. Esthetically, the tip of the nose should project at a slightly higher level than the nasal dorsum. When its forward prominence, however, is striking and attracts attention or borders on the grotesque, it approaches a caricature and must be reduced surgically. One should not confuse projection of the nasal tip with its length. A long nose is one whose dimension is unduly great in a caudal direction, whereas projection of the nose denotes an increased dimension directly forward in front of the face. Cryano de Bergerac is the classic example of this deformity. It is interesting to speculate upon the results of a modern and, we hope, successful rhinoplasty upon Cyrano de Bergerac. Certainly he would have been spared many of the arguments and adventures encountered in the play—and alas, Rostand might never have written his delightful story.

The abnormal anatomic feature that characterizes excessive nasal tip projection is an exceedingly long columella accompanied in most cases by a correspondingly large bulbous tip and an oversized dome.

In a previous article[1] I described the Safian technique for modeling the nasal tip commonly encountered. In the Safian technique, the alar cartilage is divided near the angle by a so-called cartilage-splitting incision into a medial crus and a lateral crus. The lateral crus is bounded above by the intercartilaginous incision, below by the marginal incision, and medially by the cartilage-splitting incision. Thorough dissection of the lateral crura from the overlying skin permits a direct approach to and a reduction of the cartilaginous mass in the lobule. Merely rearranging the cartilages of the tip, which is advised by so many authors, is, in my opinion, not adequate to accomplish an esthetic reduction of the prominently projected nasal tip. Cartilage must actually be removed from such a tip to produce a pleasing effect. The following is a modification of the Safian technique for modeling the nasal tip which projects too far forward.

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Technique for Lowering Extreme Nasal Tip Projection

A nasal hump when present should be removed, the cartilaginous dorsum of the nasal tip lowered[3] adequately, lateral osteotomy performed to narrow the nasal bones, the nose shortened, and the septocolumellar sutures tied *before* the tip is modeled. Not enough importance has been attached to this latter detail. It is far better to model a tip that is firm and in its final position with all septocolumellar sutures tied rather than to incise a tip that is soft and lacks firmness. In the latter situation one is compelled, after the tip has been modeled, to suture the parts together—possibly in a different relative position than one had intended originally.

Approximating septocolumellar sutures are sufficient and effective. Certainly the columella should not be overcorrected on the septum. It is advisable to use the reverse Joseph septocolumellar suture, i. e., high on the columella and low on the septum,

since this maneuver in itself tends to lower the height of the tip to a slight degree. This type of tip never shows any tendency to drop. Actually, the problem confronting us is to lower the tip enough. Neither invagination [1] nor the lip-freeing technique [2] should be employed.

In the nasal tip with *average* projection, the marginal incision should terminate on the lower border of the lateral crus near the angle at the *highest* point in the apex of the vestibule. It should not extend down on the columella, and the columella is not dissected or taken apart. I should like to emphasize that this applies to the nasal tip with average tip projection.

The tip under discussion in this paper is far different—it presents an extreme forward projection in front of the face, and therefore presents the problem of reducing the height of the columella. The marginal incision in this instance should therefore be continued down a short distance on the columella along the caudal border of the medial crus cartilage. The distance depends on the amount of projection of the tip which we wish to reduce. The greater the projection, the farther the incision extends down the columella. The average distance is approximately ¼th in.

The precise direction of the cartilage-splitting incision near the angle is the key to the entire situation and must be made with meticulous care. With the patient in the supine position, the rim of the nostril is everted with a two-pronged ball retractor

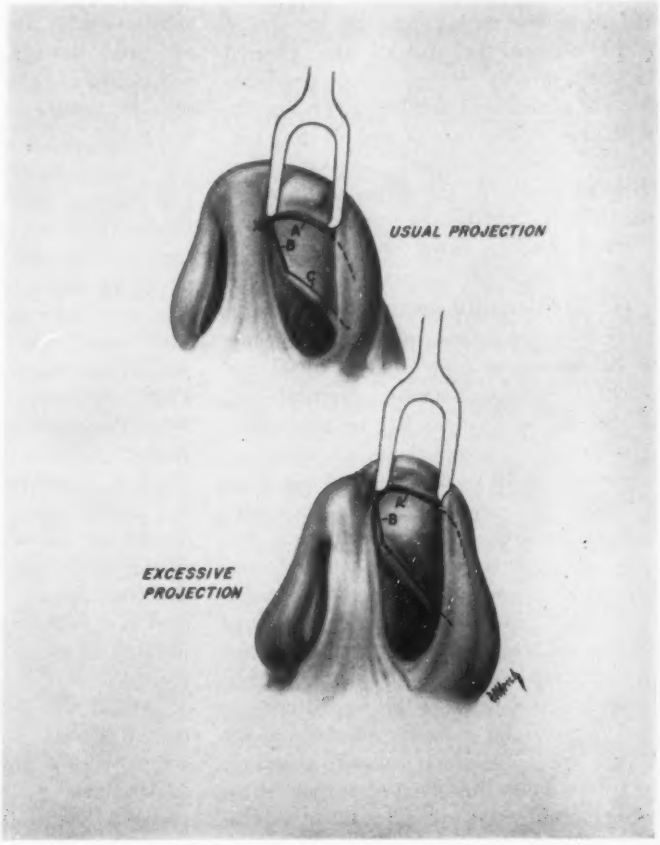


Fig. 1.—Tip with usual projection. The marginal incision (*A*) stops medially at the highest point (*X*) in the apex of the vestibule. Tip with excessive projection. The marginal incision (*A*) extends down on the columella to (*X*). (*B*), cartilage-splitting incision, and (*C*), intercartilaginous incision, are similar in the two drawings.

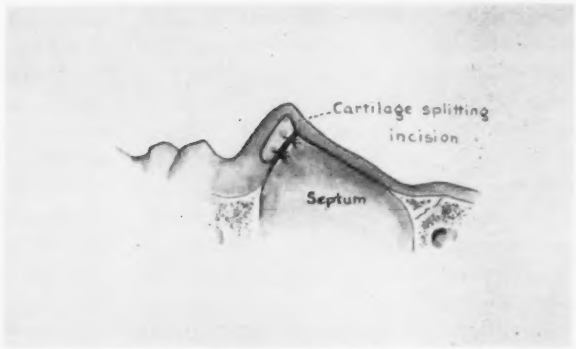


Fig. 2.—The location and direction of the cartilage-splitting incision determines the new height of the nasal tip and the creation of the slight "dip" at the supratip area.

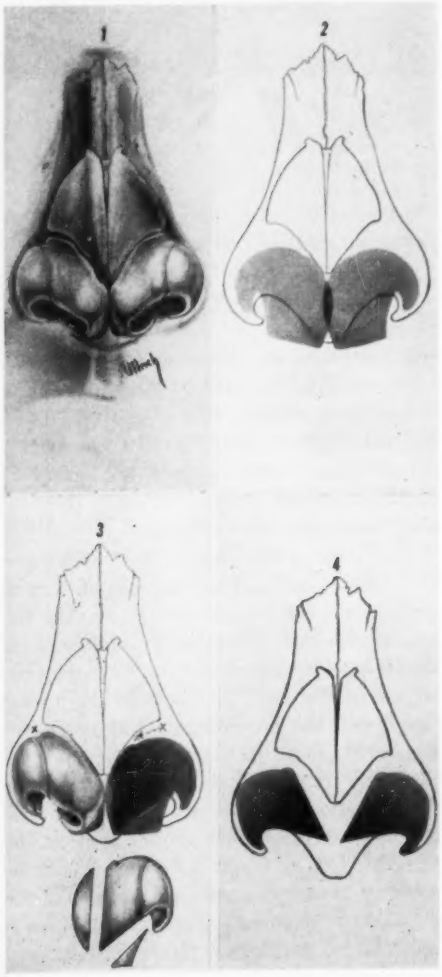
at the angle. The middle finger of the hand placed on the external skin surface of the nasal tip exerts counterpressure to bring the angle of the alar cartilage conveniently into the operative field. With a No. 15 Bard-Parker blade directed posteriorally and held at a right angle to the surface of the medial crus, an incision is made, without the use of the grooved director, directly through the vestibular skin and medial crus cartilage and columella, beginning at the medial end of the previously made marginal incision. The marginal incision in the particular tip under discussion terminates, as previously stated, a short distance down on the columella below the angle of the nasal tip.

The direction of the cartilage-splitting incision with the patient in a supine position on the operating table is in a dorsal and cephalic direction—almost vertical—and reaches the anterior surface of the septum (its dorsum) just cephalic to where the anterior and caudal septal surfaces meet. Here it joins the previously made intercartilaginous incision. The alar cartilage is thus split into a medial crus and a lateral crus. The upper, or cephalic, portion of the medial crus which contributes to the deformity of the tip and which is in part responsible for the forward projection of the tip thereby becomes detached from the medial crus by the incision and remains attached to the lateral crus. The lateral crus will ultimately be trimmed and this attached portion of the medial crus discarded. The other side of the nose is similarly treated. Nothing more need be done to the remaining portion of the medial crus. It demarcates the new height of the nasal tip. In the type of tip under discussion, it is never necessary to sew the medial crura together to obtain a narrow tip since they are always close to each other in their low position in the columella. Sewing them together might also increase to a slight degree the height of the tip—the feature which we wish to avoid.

Fine-pointed scissors introduced through the marginal incision close to the lateral crus cartilage separate the lateral crus completely from the overlying skin. The dissection should be particularly thorough at the upper lateral border of each lateral crus near the intercartilaginous incision. The lateral attachment of the lateral crus to the skin of the nasal tip remains undisturbed. This free dissection makes it possible to rotate the lateral crus medially and caudally from its position in the nasal tip to a lower position so that a small portion of its caudal and medial edges extend outside the nose beyond the rim of the nostril. The rotation of the lateral crus is made around the undisturbed lateral attachment of the lateral crus to the skin of the nasal tip. The act of rotation is a cardinal feature of the technique. It is not a mere pushing forward or sliding movement of the lateral crus. It is characterized by a true turning motion and can be thought of as though one gave a quarter-turn to a wheel, causing it to turn partially on its axis. The entire curved

surface of the dome is incorporated in the lateral crus. The vestibular skin is undisturbed and remains attached to the undersurface of the lateral crus cartilage.

Fig. 3.—*1*, Alar cartilages in preoperative position. *2*, After the cartilage-splitting incisions are made at the angles, both lateral crura are rotated outward beyond the rim of the nostril. Note that they overlap each other at the midline. *3*, The left lateral crus has been rotated outward. Note that the upper border of the left lateral crus is at a lower level after rotation than the other side. Insert reveals caudal and medial borders of the left lateral crus—excised and discarded. *4*, The trimmed lateral crura are replaced in the nasal tip adjoining the medial crura. The medial border of the lateral crura should have been drawn adjoining the medial crura at the midline. The medial crura are not shown in the drawing.



What does this rotation accomplish? The upper bulbous border of the lateral crus near the intercartilaginous incision assumes after rotation a more medial position in the nasal tip, and overlaps its fellow of the opposite side. The overlapping areas will ultimately be excised and discarded. A small portion of the original medial border of the lateral crus now extends outside the nose beyond the rim of the nostril. This protruding portion will subsequently be excised flush with the rim of the nostril and discarded.

The upper bulbous border of each lateral crus occupies a lower level in the tip after rotation than it had originally. Thus rotation of the lateral crus explains why the bulbousness of the tip seems to disappear immediately on profile view when both lateral crura are rotated outward.

The lateral crura are now ready for trimming. That portion of each crus which after rotation extends outside the nose beyond the rim of the nostril is cut straight across with straight scissors flush with the rim. The resected portion which is thus discarded is a small triangle of cartilage with its underlying vestibular skin from the original caudal and medial portions of each lateral crus. The apex of the excised triangle is situated laterally; its base medially and measures approximately $\frac{3}{8}$ in.

A crescent of cartilage with its underlying vestibular skin should now be removed with small straight scissors from the medial border of each lateral crus. Since the medial border of the lateral crus contributes to the excessive height of the nasal tip, a sufficient portion of the medial border must be excised and discarded. The incision starts near the medial edge of the marginal incision and extends slightly laterally as it approaches the upper border of the lateral crus. This crescent includes part of the dome of each side, the cephalic portion of each lateral crus which after rotation assumed a medial position in the tip, and the small cephalic portion of the medial crus which had previously been separated from

Fig. 4.—Caudal margins of the lateral crura rotated outward beyond the rim of the nostril—excised and discarded. The excised portions are never as large as depicted by the artist.

the medial crus by the cartilage-splitting incision. It is important to leave enough of the medial edge of the lateral crus at the rim of the nostril so that it reaches to but does not extend higher than the medial crus.

Sufficient cartilage therefore remains in the lateral crus along the rim of the nostril to reach barely up into the apex of the tip to the upper edge of the medial crus but not overlap it. If one is reckless or promiscuous in removing too much cartilage from the medial border of the lateral crus, notching of the margin of the tip near the apex could result.

A small rim of vestibular skin may be left attached at the medial border of each lateral crus, though this is not essential. It is my feeling that a lobule made smaller on the outside is also made smaller on the inside; and it follows therefore that a subsequently smaller lateral crus requires less vestibular skin to line its vestibular surface. It is therefore not necessary to preserve *all* of the original vestibular skin lining. The vestibular skin lining is not separated from the undersurface of the lateral crus. Atresia of the nasal vestibule does not result if ordinary precautions are followed in trimming the lateral crus cartilages.

The trimmed lateral crura meet each other at midline and adjoin the medial crura. They must not be allowed to overlap the medial crura or each other at midline. This would have the effect of increasing the projection of the tip. The height of the medial crura and the adjoining lateral crura together determine the height of the tip projection. The trimmed lateral crus at the end of the operation is triangular in shape. The apex of the triangle fits into the angle of the tip. All incisions and excisions should be equal to insure symmetry of the tip. If the decision to remove the lower end of each upper lateral cartilage is reserved to the very

Fig. 5.—Before and one year after operation. Note the preoperative forward projection of the nasal tip and dorsum, the long columella, and the bulbous tip.

last, one is invariably surprised to find that only a very small portion of these structures need be sacrificed. This is understandable, since the cephalic border of each lateral crus after rotation reaches a lower level in the tip than it had originally, and the lower end of each upper lateral cartilage acts as a substitute to fill the space vacated and created by the lower position of the cephalic border of the lateral crura.

Thus by rotating and trimming the lateral crura, and by exercising care in creating the cartilage-splitting incision, the direction, size, and shape of the lateral crura are altered.

A considerable amount of redundant fibrous tissue is sometimes present under the skin of the tip at the supratip region. When this tissue contributes to the tip deformity, its removal with tissue forceps and scissors renders heavy thick skin sometimes encountered in the tip more pliable and allows the skin to drape itself more closely to the cartilage that remains in the lobule. Such a tip appears more finely chiseled and produces a "dip" which is desirable and pleasing at the supratip region. It is not necessary or advisable to use struts, battens, or buttons of cartilage in the tip or in the columella.

Summary

An effective technique to reduce the height of an excessively forward projecting nasal tip during rhinoplasty is described.

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Treatment of Cancer of the Paranasal Sinuses and Nasal Fossa

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Treatment of cancer of the paranasal sinuses and nasal fossa is planned to destroy the tumor and rehabilitate the patient. The use of reconstructive surgery eliminates cosmetic disfigurement and prevents difficulty in mastication, swallowing, and phonation. Since most cancers in these areas are confined above the clavical until late in the course of the disease, it is possible to destroy the lesion if a diagnosis is established early and adequate therapy is administered before vital structures become involved or before the growth extends to the opposite side or to the cranium.

A communication between the mouth and nasal or antral cavities has a demoralizing effect on the patient, which can be prevented by the use of properly constructed and well-fitting prostheses. The importance of early rehabilitation of patients who have had radical surgical treatment for tumors of the jaws cannot be underestimated. Pioneer surgeons recognized this fact and used immediate prostheses to eliminate resulting deformities. According to Keen,[8] one of the early patients on whom this type of surgery was successfully performed was President Grover Cleveland, who had resection of the upper jaw and immediate insertion of a prosthesis for a malignant tumor in 1893.

Pichler [14] described a method of immediate skin grafting of the resulting cavity following resection of the upper jaw for various types of tumors and reported its use in 125 patients. I have used this technique for several years; it permits earlier use of a permanent prosthesis, prevents contracture and scarring, and shortens the period of convalescence.

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Incidence and Etiological Factors

The greatest incidence of cancer of the paranasal sinuses and nasal fossa is in persons between the ages of 50 and 70 years. The youngest patient in this group was a girl 2 years of age with fibromyxosarcoma of the left antrum (Fig. 1, *A* and *B*). It is estimated that this group of cancers accounts for approximately 1% of all human malignancies. Males are more frequently affected than females, in a ratio of two to one.

These cancers were frequently observed in the Mexican population during a five-year period when I conducted tumor clinics in the State of Coahuila, Mexico. The high incidence of these malignant tumors in Mexicans is comparable to the high incidence of malignant tumors of the nasopharynx observed in the Oriental race.

In this series of 76 patients with cancer of the paranasal sinuses and nasal fossa, no definite etiological factors were apparent. Chronic sinusitis, nasal infection, allergic rhinitis, and nasal polyps were frequently observed. Three patients with antral cancer, 28, 40, and 44 years of age, respectively, had histories of having had nasal polyps removed at frequent intervals over a period of 10 to 15 years. In each of these patients, preceding the diagnosis of malignancy, there was early recurrence of the polyps following their removal and hemorrhage became progressively more profuse.

Pathology

A number of histological types of malignant tumors are encountered in the paranasal sinuses and nasal fossa [13,15] (Table 1). The primary method of extension of these tumors is by direct invasion of surrounding tissues, and, to a less degree, by the

Fig. 1.—*A*, fibromyxosarcoma, left antrum, 2-year-old girl, which produced fullness in left cheek and obliteration of left nasolabial fold. *B*, photomicrograph demonstrating fibromyxosarcoma.

lymphatics and blood stream. Physiology of the paranasal sinuses and nasal fossa is not conducive to the embolization of cancer cells to the lymphatics and blood stream.[6] The disease, with few exceptions, i. e., cancer of the nasal septum, sphenoid and ethmoid cells, remains unilateral and does not metastasize locally or extend below the clavicle until late in its course. Two patients with carcinoma of the antrum had metastasis to the upper jugular chain of lymph nodes when first observed. Two patients developed cervical metastasis two and three years, respectively, after the primary tumor was treated, and two other patients with extensive antral cancer developed metastasis to the lymph nodes in the parotid gland area.

The maxillary sinuses are most frequently invloved [16,19,20]; there were 64 patients in this series. Two patients had carcinoma of the frontal sinuses and two others had cancer of the sphenoid and ethmoid cells. It is infrequent for antral cancer to extend to the opposite side unless it first invades the ethmoid and sphenoid cells.

A comprehensive understanding of the surgical anatomy and lymphatic pathways of the paranasal sinuses and nasal fossa is essential in the correct interpretation of early symptoms involving these structures (Fig. 2). These subjects have been presented previously [6,19] and will not be repeated here.

TABLE 1.—*Malignant Tumors of the Nasal Cavity and Paranasal Sinuses*

Type of Tumor	Nasal Cavity	Paranasal Sinuses
Squamous-cell carcinoma	2	51
Adenocarcinoma	0	5
Cylindroma	1	2
Lymphoepithelioma	1	2
Round-cell carcinoma	0	2
Fibromyxosarcoma	1	2
Fibrosarcoma	0	1
Lymphosarcoma	2	0
Malignant melanoma	1	1
Total	8	66

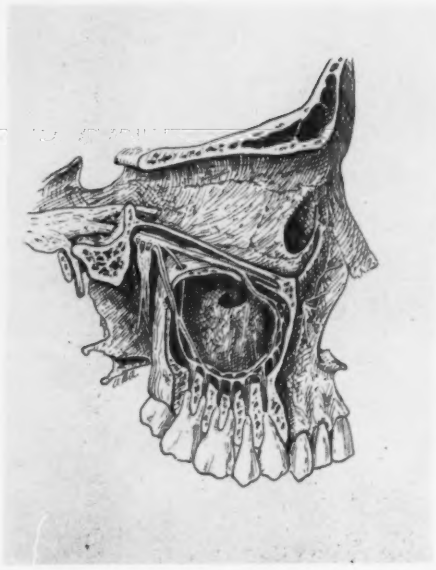


Fig. 2.—Diagrammatic representation of anatomy of right maxillary antrum. Involvement of the antrum by a neoplasm produces symptoms referable to the maxillary nerve.

Clinical Course and Symptoms

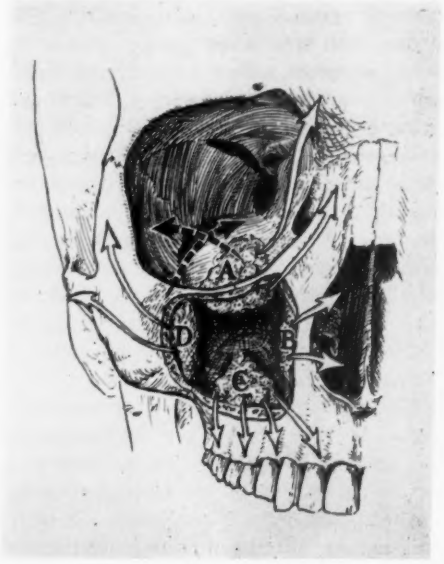
There is considerable variation in the normal course and duration of malignancies of the paranasal sinuses and nasal fossa from onset of symptoms to death if death results from this disease. This variation is due to several factors: (1) the histological type of tumor; (2) its grade, and (3) the age and general physical condition of the patient. In some patients with extremely malignant disease death may result in a period of a few months if no effective treatment is administered. On the other hand, patients affected with the histological type known as cylindroma [1,12] or a similar histological type of tumor frequently do not die from the disease for a period of years when no effective treatment is administered.

Any patient who complains of a dull, persistent pain in the face or upper jaw which is more pronounced when the patient is in a prone position may have either a benign or a malignant tumor of the paranasal sinuses. Antral cancers having their origin in the roof of the sinus invade the floor of the orbit (Fig. 3). Those located in the medial area of the roof invade the lacrimal bone and extend into the sphenoid and ethmoid cells to produce lacrimation and lateral displacement of the eye. If the tumor has its origin in the lateral area of the roof of the sinus, it extends into the temporal fossa, producing medial displacement of the eye.

Invasion of the medial wall of the antrum results in destruction of the bone with extension of the tumor into the nares, producing, unilaterally, a bloody nasal discharge which has a foul odor (Fig. 3).

Most antral cancers have their origin in the floor of the sinus in close association with the dental roots and nerves (Fig. 3). Early symptoms of growths in this location are pain, paresthesia in the region of the

Fig. 3.—Diagrammatic representation of tumors located in various areas of the maxillary antrum and their pathways of extension. Tumors having origin in the roof of the antrum (*A*) invade the floor of the orbit and extend into the sphenoid and ethmoid cells, producing lateral displacement of the eye. Tumors arising in the medial wall of the antrum (*B*) result in invasion of bone with projection into the nares, producing bloody nasal discharge. Tumors arising in the floor of the antrum (*C*) invade the tooth sockets causing loosening of teeth with pain, broadening of the upper gingiva, and ulceration of the palate. Tumors having origin in the upper lateral wall of the antrum (*D*) invade the temporal fossa and produce medial displacement of the eye.



bicuspid and molar teeth followed by loosening of these teeth, and widening of the upper gingiva. Later there is ulceration of the hard palate. When the tumor extends there is perforation of the anterior wall of the antrum with invasion of the soft tissues of the cheek and skin; the nasolabial fold is obliterated.

Any patient who develops pain over the distribution of the maxillary branch of the fifth cranial nerve should have a careful examination of the sinuses to determine if malignant disease is present. If the posterior wall of the sinus is involved, frequently there is invasion of the pterygomaxillary fossa resulting in pain referred to the angle of the mandible and ear; trismus will be present (Fig. 3). Hara [4] reports two patients with cancer of the antrum in whom the pterygoid fossa was invaded; both patients had undergone intracranial operations on the maxillary branch of the fifth cranial nerve because it was not realized that these patients had antral cancer. Frequently patients are observed with antral cancer or primary tumors of the maxilla or buccal mucous membrane whose chief complaint is that their dentures have ceased to fit.[5,7,20]

Tumors located in the area of the sphenoid and ethmoid cells produce nasal obstruction, bloody nasal discharge, and lacrimation, and frequently there is secondary sinusitis produced by a blockage of the osteum without proper drainage.

The most frequent symptoms of either benign or malignant tumors of the nasal fossa are unilateral nasal obstruction, nasal discharge, and epistaxis.[1] Tumors in this location produce broadening of the bridge of the nose with obliteration of the nasolabial fold; later the skin over the nose becomes ulcerated. There may be lateral displacement of the globes with exophthalmos when the orbits are invaded.

Differential Diagnosis

Benign cysts and tumors involving the paranasal sinuses and nasal fossa frequently produce symptoms similar to those of malignant tumors of these structures, and a differentiation must be made. Unilateral or bilateral nasal polyps are one of the most frequent diseases producing nasal obstruction and may occur at any age.[11,13] Rhinoscleromas, nasopharyngeal fibromas, papillomas, and adenomas are rare. Unilateral chronic sinusitis, osteomas, ameloblastomas,[21] odontogenic cysts, and mucoceles occur in the antrum and frontal sinuses and may simulate malignant tumors. Eight patients with mucoceles were encountered in the period during which this group of malignant tumors of the paranasal sinuses and nasal fossa was treated. The symptoms of mucocele are intermittent attacks of pain in the region of the frontal or maxillary sinuses, pain in the premolar and molar teeth, anesthesia and paresthesia of the teeth on the involved side, pressure necrosis of one or more sinus walls, and, not infrequently, diplopia. One patient who had an early antral cancer involving the roof of the antrum also had a large mucocele of the frontal sinus producing destruction of the roof of the orbit and lateral displacement of the globe.

Meningioma, when arising from the sphenoid ridge, may invade the orbit, zygomatic fossa, or paranasal sinuses. A previous report [20] demonstrates the apparent benign nature of these lesions in their early stages and the fact that gratifying results can be obtained by surgical measures if definitive therapy is utilized in the early stage of the disease. The extent of the meningioma is determined by roentgenographic studies which will demonstrate if there is extension of the tumor from the cranial cavity into the orbital floor or pterygoid fossa.

Roentgenographic studies have only limited value in the diagnosis of early carcinoma of the paranasal sinuses. Since most malignant tumors of the paranasal sinuses and nasal fossa have their origin in the lining membrane of these cavities, and since early carcinoma is a soft-tissue growth, the shadow produced on a roentgenogram is similar in appearance to one produced by an

inflammatory process.[18] The first definite evidence of malignant tumor by roentgenogram is bone destruction produced by bone invasion of the sinus wall whereas the surrounding bony tissue appears normal. As the disease progresses and the wall is completely destroyed, there is a sharp contrast between normal bone and bone which has been destroyed (which is characteristic of neoplastic involvement). Because there is natural overlapping of bone shadows in this anatomical area, early malignant disease may not be detected radiologically unless great care is exercised by the radiologist. Of the 68 patients with malignant tumors of the paranasal sinuses, 50 demonstrated destruction of one or more sinus walls by radiographic examination. Tomographs have proved of value in these cases, in addition to standard x-ray films.

In carcinoma of the paranasal sinuses or nasal fossa, a diagnosis is established by histological study of representative tumor tissue, and the type of tumor is determined. The tissue to be examined is removed either through a Caldwell-Luc incision or by aspiration biopsy, using a Turkel needle, which permits a core of tissue to be obtained. When a Caldwell-Luc incision is made, care is exercised to obtain representative tissue, not necrotic tissue from the surface of the tumor. If aspiration biopsy is used, the sinus is entered by inserting a Turkel needle through the gingival sulcus or directly through the skin of the cheek if the anterior wall of the sinus has been destroyed. Not infrequently, following biopsy through a Caldwell-Luc incision, there is active bleeding, which is controlled with absorbable gelatin sponge (Gelfoam) and a pressure dressing.

Treatment

Spontaneous cure of patients with malignant disease, although long a favorite theory of "arm-chair" students of oncology, actually has not been observed by those who treat patients with malignant disease. In general, all patients with carcinoma of the paranasal sinuses and nasal fossa are treated with radiation and/or surgery. A careful examination is made of each patient to determine if there is extension of the tumor to the opposite side or to the cranium and whether or not there is local or distant metastases. Radiosensitive tumors, i. e., anaplastic epidermoid carcinoma, transitional-cell epithelioma, and lymphosarcoma, or tumors occuring in patients with advanced disease, or those in patients whose general physical condition will not permit radical surgery, are treated with radiation alone.

Treatment of Cancer of the Paranasal Sinuses.—It has been approximately 100 years since the first successful operation was performed for cancer of the maxillary antrum; however, even today, with our refinements in diagnostic procedures, irradiation and surgical techniques, improvements in anesthesia, the use of antibiotic and blood therapy, as well as the use of well-made prostheses, the end-result of treatment of cancer of these structures is not entirely satisfactory. This field of research has not lost its urgency to reduce the mortality associated with the disease.

It is my practice to treat patients with antral cancer with radiation and/or surgery after a careful evaluation of the patient is made. Patients of advanced age whose general physical condition will not permit radical surgery, and those in the advanced stage of the disease, are treated with radiation alone. In 20 of the 68 patients with cancer of the paranasal sinuses, the disease was advanced to the stage that only radiation was administered. Patients whose physical condition will permit radical surgery are treated with preoperative radiation; the total dose is less than that administered to patients who are treated with radiation only.

Radiation therapy has undergone many refinements and may be administered in higher doses than formerly with present-day application of precision techniques. It must be appreciated that because of the anatomical formation of the maxillary antrum, it is difficult to administer a homogeneous dose

of radiation throughout the tumor-bearing area.[2] Irradiation, with conventional voltage using cross-fire technique, may result in overtreatment of one area and undertreatment of other areas and permit viable cancer cells to remain. Also, when given in sufficient quantities to destroy cancer contained in a box of bone such as the antrum, irradiation frequently results in a residue of complications, i. e., necrosis, radio-osteonecrosis, sclerosis, obliterative endarteritis, or chronic osteomyelitis, bringing about permanent changes in soft as well as osseous tissue. These complications, which are extremely painful and debilitating, may require surgical treatment and antibiotic therapy.

The use of cobalt teletherapy, or supervoltage radiation, which eliminates the use of conventional cross-fire fields, may permit more adequate irradiation of antral carcinoma. A cancericidal dose of 5000 to 6000 r, delivered over a period of four to six weeks should be attended with less irradiation morbidity. Devine and associates[2] caution that, since this method of irradiation is relatively new, time will be required to determine long-range results from its use. These authors also stress that, unit for unit, supervoltage therapy is not, biologically, as effective as conventional irradiation; hence, when supervoltage is used, there must be an increase in the dose by one-third over that used in conventional therapy.

Radiation is administered through three ports, varying in size from 4 to 6 cm. If the orbit or ethmoid and/or sphenoid cells are involved, or if there is extension of the tumor into the pterygoid fossa, a fourth or fifth port (if necessary) is used: one through the zygoma just above the lateral port, and one straight through the orbit from the front. The dose varies from 2000 to 3000 r through each port. Treatment factors are as follows: 260 kv., 15 ma., 0.5 mm. copper plus 1 mm. aluminum filter, 50 cm. target-skin distance. Treatments are administered daily in doses of 300 r, which result in epithelialitis, desquamation, and occasionally, blistering; these complications disappear after a period of four to six weeks.

Surgical Treatment of Cancer of the Paranasal Sinuses.—Conservative surgery does not permit removal of sufficient tissue to effect a cure for patients with cancer of the paranasal sinuses.[5,17] It is necessary to resect the superior maxilla and frequently exenterate the orbital contents. If the lesion is limited to the floor of the antrum, and if there is no evidence of invasion of the orbital floor, the resection is limited to the superior maxilla below the orbit. However, if there is extension of the lesion to involve the orbital floor, or if the origin of the tumor is in the roof of the antrum or frontal sinuses, it is necessary to exenterate the orbital contents. It was necessary to resect the orbital contents in 30 of the 44 patients in this series who had radiation therapy followed by radical surgery. When the orbital contents are to be exenterated, the question regarding the resulting disfigurement (loss of one eye) is present. Patients can be trained, after removal of one eye, to depend on the other. Complications resulting from the loss of one eye are totally outweighed by the increased protection allowed by a more extensive operation for cancer involving the orbital contents. The defect produced permits more thorough follow-up examinations of the operative site. If persistent or recurrent tumor is present, postoperative irradiation is administered to such areas directly through a cone. The patients in this series in whom exenteration of the orbital contents was essential have not suffered to any degree from the loss of an eye. They are more content in the knowledge that they have a better opportunity of being permanently well than if the eye had not been removed.

Contraindications to operability are (1) invasion of the base of the skull; (2) bilateral involvement demonstrated radiologically in cancer of the frontal sinuses, ethmoid and sphenoid cells, and those cases in which the medial wall of the antrum is involved and the lesion has extended to the opposite side (Even though a lesion involving the medial wall of the antrum which has extended to the opposite side could be

adequately removed, it would be impossible to correct the resulting defect.[6]); (3) involvement of the wall of the nasopharynx; (4) metastases below the clavicle; (5) patients of advanced age with associated debility. Only two patients in this group had metastases to the regional lymph nodes when first observed; these patients had radical neck dissection in addition to resection of the superior maxilla, and one had exenteration of the orbit.

The operative procedure is carried out through a Fergusson incision, which reveals the extent of the tumor and permits exploration of surrounding structures, i. e., nasal cavity, orbit, nasopharynx, pterygoid fossa, ethmoid and sphenoid cells. The patient is placed on the operating table on his back with the head of the table lowered. The neck is slightly extended to facilitate drainage of secretions and blood from the respiratory tree. Intratracheal anesthesia, with a tube inserted through the mouth on the uninvolved side, has proved to be the anesthetic of choice. Thiopental (Pentothal) sodium, oxygen, and nitrous oxide are usually used throughout the operation, thereby permitting use of an electrosurgical current. The pharynx is carefully packed off with a moist pack. The external carotid artery and its branches are ligated on the involved side at the beginning of the operative procedure to reduce bleeding during and after operation. An incision is made with an electrosurgical current in the midline of the upper lip to a point beneath the columella of the nose; it is extended around the ala of the nose in the nasolabial fold to the level of the inner canthus of the eye (if the eye is to be preserved), then is extended laterally just above the infraorbital ridge, with the concavity upward, across the malar bone to a point above the zygomatic process (Fig. 4, *A*). The incision is made through the thickness of the lip and cheek to the bone throughout its entire length except in front of the ear. The infraorbital branch of the facial nerve which follows the upper border of the zygomatic process, is preserved, if possible, to prevent paralysis of the lower

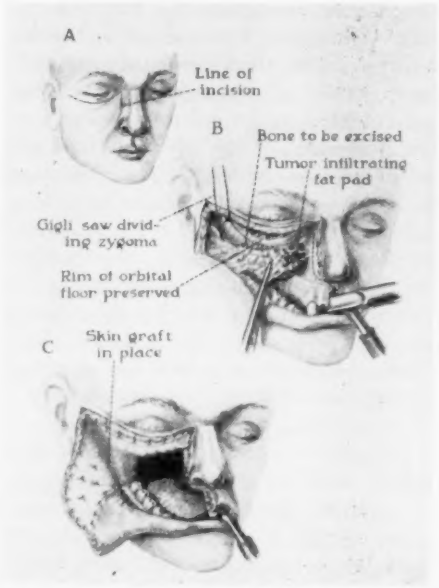


Fig. 4.—*A*, diagram demonstrating Fergusson incision through which reflection of the cheek and resection of the superior maxilla are carried out for cancer of the paranasal sinuses. *B*, the cheek flap is turned back demonstrating tumor infiltrating the anterior antral wall with involvement of subcutaneous tissue. The broken line demonstrates where bone incision is made (with hammer and chisel) through the middle of the upper jaw to the floor of the nose, across the nasal bone; the infraorbital ridge is preserved, and the zygoma is severed with a Gigli saw. *C*, the superior maxilla has been resected. A split-thickness skin graft covers all raw surfaces of the cheek, masseter muscle, and pterygoid fossa.

half of the orbicularis muscle; if exenteration of the orbit is performed, this branch of the facial nerve need not be preserved. The skin and soft tissues of the cheek are reflected to leave the subcutaneous fat over the maxilla (Fig. 4, *B*). If there is involvement of the skin, it is removed also and the resulting defect is covered with a pedicle flap from the neck or forehead at a later date.

The mouth is opened widely and the central incisor tooth on the involved side, if present, is extracted. The mucous membrane in the gingivolabial sulcus is severed with a scalpel. An incision with an electrosurgical current is made in the midline of the hard palate and extended backward to

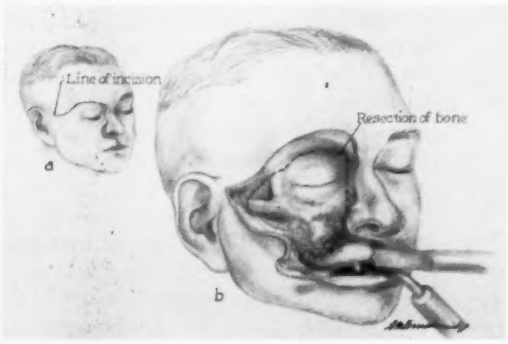


Fig. 5.—(*a*) Technique used for resection of maxilla and orbital contents. Incision is made through midline of the lip to columella of the nose, around the ala of the nose in the nasolabial fold to the eyebrow. The cheek flap is turned back. (*b*) With hammer and chisel, incision is made through the hard palate and maxilla. The zygoma is severed with a Gigli saw. (Reproduced with permission from Hendrick, J. W.: *Surg. Gynec. & Obst.* 102:322-330, 1956.)

the soft palate, across the posterior edge of the hard palate to the superior alveolar tuberosity to meet the incision in the gingivolabial sulcus.[29] The incision is completed with a chisel and hammer through the midline of the palate to its posterior border and carried through the juncture of the superior maxilla and nasal bones. The zygoma and malar bone are examined, and any involved portion of the zygoma is removed with a Gigli saw. If the orbit and orbital contents are not involved, the infraorbital ridge is preserved, which prevents double vision and loss of function of the eye; the superior maxilla is now readily removed with heavy bone forceps. There is usually brisk bleeding at this point of the operation; the smaller vessels are clamped and coagulated with an electrocoagulator, and the larger vessels are ligated with fine chromic absorbable surgical suture. Any area of the sphenoid or ethmoid cells or pterygoid fossa found to be involved by cancer (after careful inspection of these structures) is destroyed with an electrocoagulator. The involved tissue is coagulated, curreted, and recoagulated until normal-appearing tissue is found. Remnants of involved bone are removed with a rongeur and treated with an electrocoagulator.

Involvement of the infraorbital ridge and orbital contents makes exenteration of the eye imperative. In patients in whom exenteration of the orbit is to be performed, the incision is extended to the level of the eyebrow, then laterally along the supraorbital ridge above the zygomatic process. In patients in whom the zygoma is involved, the incision is extended to the anterior surface of the ear and downward in front of the auditory canal to the level of the lobe, which permits exposure of the entire zygoma and surrounding tissues (Fig. 5).

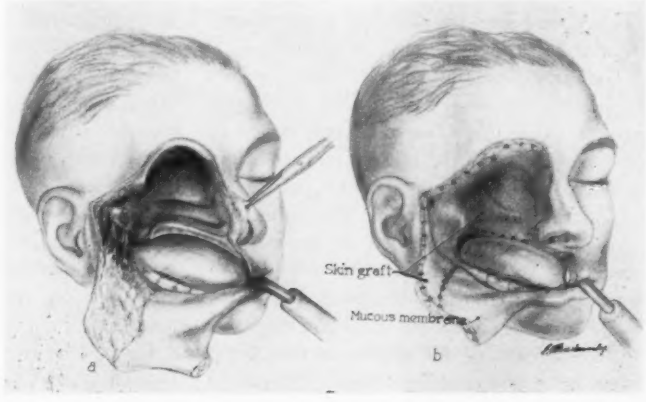


Fig. 6.—(*a*) The superior maxilla and orbital contents have been removed. (*b*) The raw surface of the cheek flap and orbit are covered with a split-thickness skin graft; the cheek flap is resutured in place. (Reproduced with permission from Hendrick, J. W.: *Surg. Gynec. & Obst.* 102:322-330, 1956.)

The use of a split-thickness skin graft, large enough to cover all raw surfaces of the maxillary cavity, cheek, and orbit, greatly improves the results of this operative procedure[14] (Fig. 6, *a*, *b*). The graft is trimmed and sutured in place with No. 0 chromic absorbable surgical sutures. A few absorbable sutures are placed throughout the graft to obliterate dead space beneath it. The use of the split-thickness skin graft eliminates former complications of the operative procedure by covering the large raw surface which otherwise would be covered with dry secretions, by hastening the convalescence period, by decreasing scarring and contracture, and by permitting a permanent prosthesis to be worn within a few days after operation.

It is my practice to treat any area which may contain residual cancer with radium needles or tubes in rubber cots; radium used in this manner can be placed in areas where it is most desired. The total dose is calculated to give 6000 to 10,000 r at 1 cm. depth. The radium tubes, or needles, are attached to long silk sutures which are brought out through the most convenient angle of the wound.

The resulting cavity is covered firmly with iodoform gauze, which holds the skin graft snugly in place and prevents postoperative oozing. The cheek and lip flap are sutured back in their normal beds. The muscles and subcutaneous tissues are sutured with No. 0 chromic absorbable surgical sutures; the skin is closed with interrupted sutures of black silk. A snug pressure dressing is placed over the cheek and held with an Ace bandage.

Carcinoma of the frontal sinuses is not frequently encountered. There were two patients with this disease in this series of cases, and in each the disease had its origin in the floor of the sinus. In one patient the medial area of the floor of the sinus was involved, and in the second the lateral wall was involved. After adequate biopsy, preoperative radiation is administered with the beam directed over the tumor, using two ports. In each of the above-mentioned patients it was necessary to remove the orbital contents, but it was not necessary to resect the floor of the maxilla. In one patient the roof of the anterior fossa and cribriform plate were destroyed. Both of these patients are clinically free of the disease, one three years and the second one year after treatment.

When the malignancy has its origin in the ethmoid or sphenoid cells, surgical exposure of the tumor is preferred before any preoperative radiation is administered. All tumor-bearing tissue is electrocoagulated, curetted, and recoagulated until normal-appearing tissue remains. Radium, in the form of plaques, is used, giving a total dose of 6000 to 10,000 r. The two patients who had cancer arising in the sphenoid or ethmoid cells died from intracranial invasion within a period of two years.

Treatment of Cancer of the Nasal Fossa.

Small squamous-cell carcinomas of the mucocutaneous junction of the columella are treated with radiation by the same technique used for squamous-cell carcinoma of the skin, or they may be treated by direct application of radium tubes placed against the growth with a small lead plate between the tubes and the ala of the nose to protect the latter.[10] Radiosensitive tumors of the nasal fossa are treated with radiation, using 260 kv., 15 ma., 0.5 to 1.5 mm. copper, and 1 mm. aluminum filters, 50 cm. target-skin distance. Daily treatments are administered in varying amounts, depending on the size of the field, to give a total of 4000 to 5000 r. If necessary, two or three ports are used. The uninvolved, surrounding areas are protected by a sheet of lead 0.25 cm. in thickness. Some extensive, well-differentiated carcinomas of the nasal fossa which are not considered to be radiosensitive are first treated with radiation, using 3000 to 4000 r, administered over a period of three to four weeks. The patient is observed at the end of six weeks, and any remaining tumor is removed surgically through a lateral rhinotomy incision, which has given satisfactory results.

Cancers of the turbinate bones and nasal septum which are radioresistant are treated

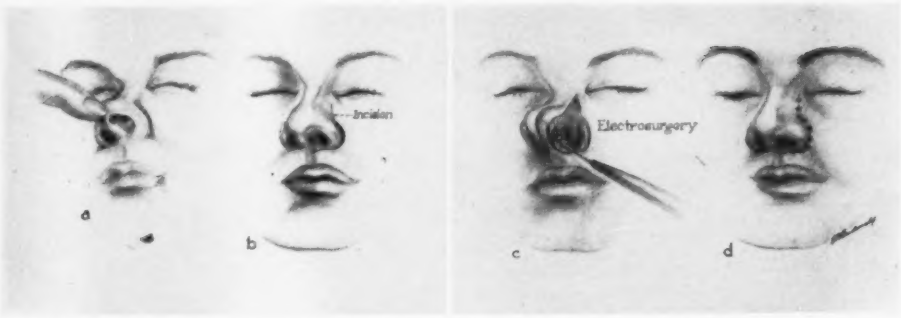
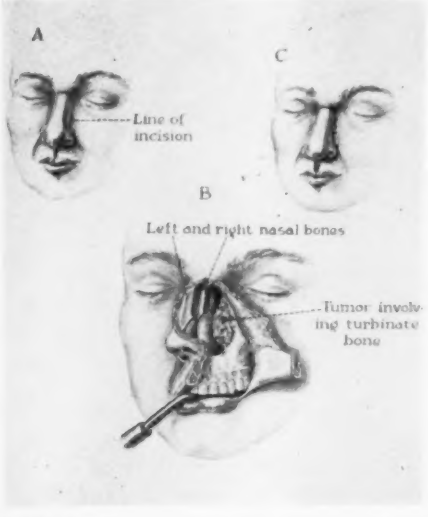


Fig. 7.—(*a*) Diagrammatic representation of a tumor involving the turbinate bones or nasal septum. (*b*) A lateral rhinotomy incision is made through the upper lip and extended as high as necessary in the nasolabial fold, the ala of the nose is turned medially, permitting small tumors to be removed from the septum and turbinates. (*c*) The tumor is thoroughly destroyed with electrocoagulation; the muscles and subcutaneous tissues are closed with fine interrupted chromic catgut sutures. (*d*) The skin is closed with interrupted mattress sutures of fine silk. (Reproduced with permission from Hendrick, J. W.: *Surg. Gynec. & Obst.* 102:322-330, 1956.)

with electrosurgery and scalpel surgery through a lateral rhinotomy incision (Fig. 7, *a*). The incision is begun in the floor of the nares on the involved side and extended around the ala of the nose in the nasolabial fold to the inner canthus of the eye (Fig. 7, *b*). The tumor is thoroughly destroyed with electrocoagulation; the muscles and subcutaneous tissues are closed with fine interrupted chromic absorbable surgical sutures. The skin is closed with interrupted mattress sutures of fine silk (Fig. 7, *c*, *d*). More extensive growths are exposed by an incision made in the midline of the upper lip and extended in the same line as the above incision to the height desired; this further extension of the incision permits the cheek to be turned back to determine if there is invasion of the antrum or involvement of the opposite nares (Fig. 8 *A*, *B*). Tumors involving the posterior area of the turbinate bones and nasal septum, or those extending into the antrum, are well managed by the use of this technique; the cosmetic results are satisfactory. The muscles and subcutaneous tissues are closed with fine interrupted chromic absorbable surgical sutures; the skin is closed with interrupted mattress sutures of silk.

Fig. 8.—*A*, *B*, and *C*, diagrammatic representation of method of removal of more extensive tumors of the nasal fossa. An incision is made through the midline of the upper lip to the columella of the nose; it is extended around the ala in the nasolabial fold to the height desired, which permits careful inspection of a tumor involving the posterior nasal septum, posterior area of the turbinates, or floor of the nares. If there is extension of the tumor into the antrum, the cheek can be reflected and the tumor thoroughly removed with electrocoagulation. The muscles and subcutaneous tissues are closed with interrupted fine chromic absorbable surgical sutures; the skin is closed with interrupted mattress sutures of silk. This procedure gives good cosmetic results.



Radical Neck Dissection.—Radical neck dissection is not included with resection of the superior maxilla for cancer of the paranasal sinuses unless there is a definite evidence of metastasis. Four patients had

TABLE 2.—*Metastases from Cancer of the Paranasal Sinuses in Six Patients*

No. of Patients	Time of Metastasis	Site of Metastasis	Type of Therapy	Results
1	When first observed	Jugular chain of lymph nodes	Radical neck dissection	Living, free of disease, 3 yr.
1	When first observed	Jugular chain of lymph nodes	Radical neck dissection	Living free of disease, 5 yr.
1	2 yr. after resection of maxilla	Jugular chain of lymph nodes	Radical neck dissection	Living, free of disease, 3 yr.
1	3 yr. after resection of maxilla	Jugular chain of lymph nodes	Radical neck dissection	Living, free of disease, 5 yr.
1	2 yr. after resection of maxilla	Parotid lymph nodes	Implantation of radium needles	Expired within 1 yr.
1	3 yr. after resection of maxilla	Parotid lymph nodes	Implantation of radium needles	Living, free of disease, 4 yr.

radical neck dissection: two who had metastases to the upper jugular chain of lymph nodes when first observed and two others who developed metastases two and three years, respectively, after resection of the superior maxilla. All four patients are living, with no clinical evidence of recurrence of their disease, one after two years, one after three years, and two after five years (Table 2).

Postoperative Care.—Postoperative care is carried out by placing the patient on the involved side in his hospital bed; he is encouraged to be out of bed the following day. Edema of soft tissues is reduced by warm saline dressings. Patients having resection of the superior maxilla have difficulty swallowing for a period of several days; they are administered nasal feedings consisting of a high-caloric and high-vitamin diet, 3000 to 4000 calories daily, with use of an ordinary No. 18 or 20 French urethral catheter,[11] which the patient is taught to insert himself. The feedings are begun 24 hours after surgery and continued as long as necessary. When the wound is dressed, the use of hydrogen peroxide permits the iodoform packs to be readily removed. The wound is irrigated with a warm solution of sodium bicarbonate and sodium chloride, and the cavity is repacked with iodoform gauze.

Radium tubes or needles, if used, are removed with the iodoform gauze after the desired dose of irradiation has been administered. The gauze pack is removed after five or six days when radium is not used.

It has been my practice to have a prosthesis prepared by a dental surgeon prior to the time of operation, and this prothesis is inserted as soon as the local wound will permit. This affords the patient a means of eating, drinking, and speaking without difficulty, and adds greatly to his morale.

Comment

Unfortunately, early diagnosis is not always made in carcinoma of the paranasal sinuses and nasal fossa. In 40 patients with cancer of the paranasal sinuses and in 4 with cancer of the nasal fossa, the disease was advanced when a definitive diagnosis was established. Irradiation, only, was used in 20 patients with antral cancer because of the presence of one or more contraindications to surgery, i. e., advanced age, additional complicating disease, generally poor physical condition, radiological evidence of extension of the disease to the base of the skull or into the pterygoid fossa, nasopharynx, or soft tissues, bilateral spread of the disease, evidence of generalized metastasis, or refusal by the patient to undergo surgery. Of the patients treated with radiation only 10 have been followed from four to seven years: 7 died from recurrent cancer; 1 died from an unrelated disease; 2 are living and clinically free of cancer but have complications resulting from radiation therapy. The other 10 of the 20 patients who were treated only with radiation have

TABLE 3.—*Results of Treatment of Sixty-Eight Patients with Cancer of the Paranasal Sinuses*

No. of Patients	Type of Therapy	Died from Cancer	Died from Unrelated Disease	Lost to Follow-Up	Living, Free of Disease 4-7 Yr.
24	Irradiation & surgery	2	2	1	19
10	Irradiation only	7	1	0	2
25	Irradiation & surgery	(Followed 3 mo. to 4 yr., no data compiled)			
5	Irradiation only	(Followed 3 mo. to 4 yr., no data compiled)			
2	(Refused all therapy)				
2	(Disease too advanced to warrant therapy)				
Total 68		9	3	1	21

been followed from a few months to three years, and no statistical data have been compiled on them.

Preoperative irradiation is used for all patients with cancer of the paranasal sinuses in whom the disease is found to be operable for the reasons that (1) the growths are active when first observed; (2) the lymphatic channels and adjacent tissues are being invaded, and (3) irradiation destroys and devitalizes a substantial amount of the tumor growth and lessens dissemination of malignant cells during the operative procedure.[5] It is thought to also obstruct the lymphatic channels. In three patients who had radical resection of the maxilla and/or orbital contents following preoperative irradiation, no viable cancer cells were reported in the pathological specimen.

The operative procedure is performed from three to six weeks after completion of radiation therapy. Delay in healing of tissues after administration of preoperative radiation has not been observed in these patients.

Forty-four patients with cancer of the paranasal sinuses had irradiation followed by radical surgery. Twenty-one of these patients have been followed from four to seven years; one has been lost to follow-up; two have died from unrelated disease; two have died from recurrent cancer, and nineteen are living and have been clinically free of their disease from four to seven years (Table 3).

The other 23 patients who had preoperative irradiation followed by radical surgery have been followed from three months to four years, and no data have been compiled on any patient treated under three years.

There were eight patients with cancer of the nasal fossa; four who were treated with radiation died from one to three years after they were first observed. The other four patients were treated with lateral rhinotomy incision with thorough removal of the tumor by electrosurgery; these have been followed from four to seven years and are clinically free of their disease (Table 4).

The average postoperative hospital residence for patients who underwent surgery was 11 days; there was no hospital mortality.

Larsson and Maartensson,[9] reporting on 334 patients with carcinoma of the paranasal sinuses from the Radiumhemmet and Karolinska Hospital in Stockholm, used irradiation, alone, on 206 patients whose cancers were principally inoperable. Combined irradiation and surgery were used on 128 patients. These authors reported a five-year

TABLE 4.—*Results of Treatment of Eight Patients with Cancer of the Nasal Fossa*

Type of Tumor	No. of Patients	Stage of Disease	Type of Therapy	Patients Living, Free of Disease 3-7 Yr.
Squamous-cell carcinoma	2	Early	Surgery	2
Cylindroma	1	Early	Surgery	1
Malignant melanoma	1	Early	Surgery	1
Lymphoepithelioma	1	Advanced	Irradiation	0
Fibromyxosarcoma	1	Advanced	Irradiation	0
Lymphosarcoma	2	Advanced	Irradiation	0
Total	8			4

cure rate with combined irradiation and surgery of 45%; with the use of irradiation alone, the five-year cure rate was 12%. In their entire series, with the use of all methods of therapy, the five-year cure rate was 23%. During the treatment period lymph-node metastases occurred in 23% of their patients and generalized metastases were encountered in 10%. Their results were unfavorable for patients with lymph-node metastases on admission.

When a diagnosis is established early, the method of treatment of cancer of the paranasal sinuses which gives the best curative results and that which is most widely used at this time is radical surgery with preoperative and/or postoperative irradiation.

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Audiology Section

JOSEPH SATALOFF, ASSOCIATE EDITOR

Clinical Observations on Excessive Threshold Adaptation

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In 1955, Lierle and Reger [1] reported an unusual finding in connection with their extensive studies of threshold tracings obtained with the Békésy-type audiometer. When subjects were asked simply to trace threshold for a fixed frequency over a 20-minute period, they found that the tracings remained relatively constant in two cases of end-organ lesion, but dropped markedly in one patient with a subsequently confirmed eighth-nerve tumor. In other words, in the case of end-organ lesion, the intensity level required to maintain a threshold response did not change appreciably over the 20-minute tracing period. In the case of eighth-nerve lesion, however, the patient rapidly required more and more intensity to keep the tone at threshold. Similar results were subsequently reported by Kos.[2]

This finding is, of course, exactly opposite to the earlier observations of Dix and Hood.[3] According to these investigators, the threshold "adaptation" exemplified by the gradual decline in the threshold tracing over time characterizes and, indeed may be attributed to, certain features of cochlear as opposed to retrocochlear pathology.

In view of these seemingly conflicting reports, we think it is important to report that we have confirmed the findings of Lierle and Reger in two cases of acoustic neurinoma. Conversely, the threshold tracings we have obtained in presumably cochlear losses invariably fail to support the position of Dix and Hood.

We have selected, for illustrative purposes, two cases exemplifying this distinction. Figure 1a shows the preoperative audiogram of a 45-year-old white man with a unilateral loss of the sensorineural type. Speech reception threshold and discrimination of PB words were within normal limits on the right ear, but, on the left ear, the patient was unable to understand speech of any kind. Subsequent surgical intervention revealed a relatively large acoustic neurinoma in the left cerebellopontine angle, extending from the tentorium above to the

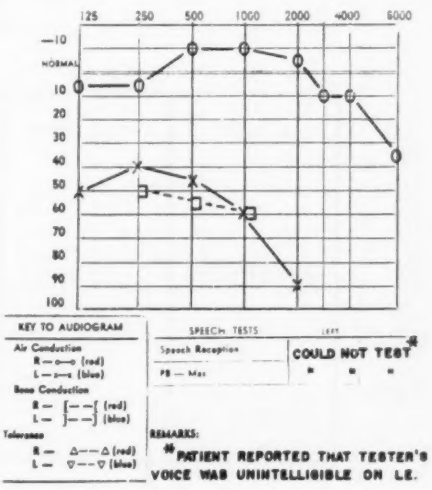

Fig. 1a.—Patient 1.

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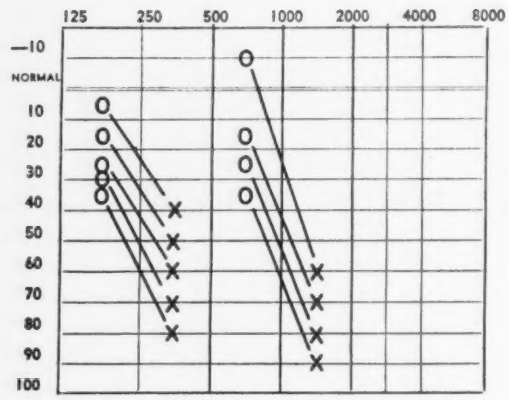

Fig. 1b.—Patient 1.

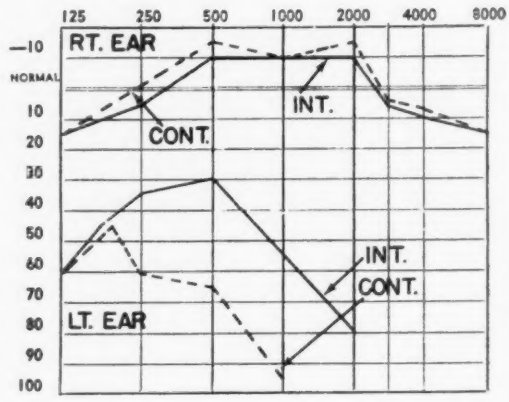

Fig. 1c.—Patient 1.

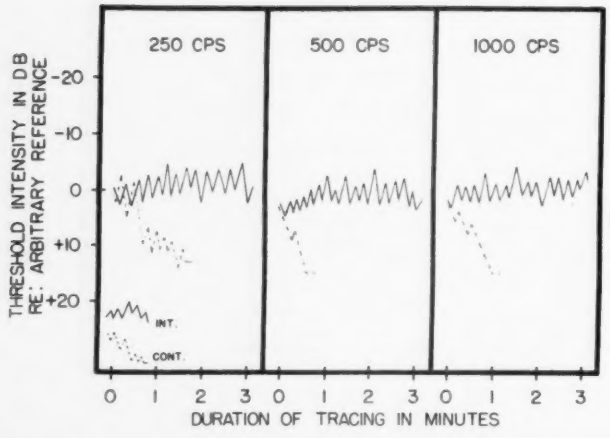

Fig. 1d.—Patient 1. Test ear: left.

foramen magnum below and extending laterally into the internal auditory meatus.

Figures 1b through 1d summarize the results of other special tests performed preoperatively. Figure 1b shows the results of alternate binaural loudness balances at 250 and 1000 cps. There is little, if any, evidence of loudness recruitment on the left ear. Figure 1c shows the hearing loss as measured with the Békésy-type audiometer (Grason-Stadler, type E-800). The solid curve represents the audiogram obtained with a pulsing stimulus which was interrupted three times per second, while the dashed curve represents the audiogram obtained with a noninterrupted, or continuous, stimulus.

On the right, or normal, ear the two kinds of tracing stimuli, continuous and interrupted, yield essentially equivalent thresholds, but on the left, or impaired, ear an interesting pattern emerges. At very low frequencies, the two thresholds overlap. At about 170 cps, however, they diverge sharply. Above this frequency, the patient requires considerably more intensity to maintain the continuous tone at threshold than to maintain the interrupted tone at threshold. At 1000 cps, the difference reaches the extreme magnitude of 40 db.

Figure 1d shows the same phenomenon in another way. Here the patient traced threshold for a tone of fixed frequency for three minutes. While he was listening to the interrupted tracing tone, the threshold remained relatively stable over the three-minute period. When the tracing tone was continuous, however, the tracings dropped rapidly, indicating that the patient required progressively more and more intensity in order to keep the continuous tone audible. Of particular interest is the rapidity and severity of this drop. At 1000 cps, for example, the tracing dropped 35 db., to the limit of the equipment, in less than 30 seconds. Even at 250 cps a drop of 35 db. occurred in less than 90 seconds.

We have observed this same pattern of results in a second case of surgically confirmed acoustic neurinoma. Again, in this case, loudness recruitment was absent on the affected ear, and, again, the patient was unable to understand any kind of speech delivered to the impaired ear.

The marked difference between the threshold tracings of continuous as opposed to interrupted stimuli observed in these two cases of acoustic neurinoma contrasts sharply with the behavior in cases of end-organ lesion. Figure 2a, for example, shows the conventional audiogram of a 34-year-old white man with a unilateral loss, clinically diagnosed as due to Ménière's disease. Figure 2b shows the result of alternate binaural loudness balances establishing the presence of loudness recruitment on the impaired right ear. Administration of a standard PB-word list by live voice at a level of 40 db. above the spondee threshold yielded a discrimination score of 54% on the right ear.

Figure 2c shows the audiogram for both interrupted and continuous stimuli as measured on the Békésy-type audiometer. Here, the two types of stimuli result in nearly identical thresholds on both the normal and the impaired ear. The continuous tone threshold drops somewhat below the interrupted tone threshold at frequencies above 1000 cps on the impaired ear, but the difference is slight in comparison with the marked disparity observed in Figure 1c.

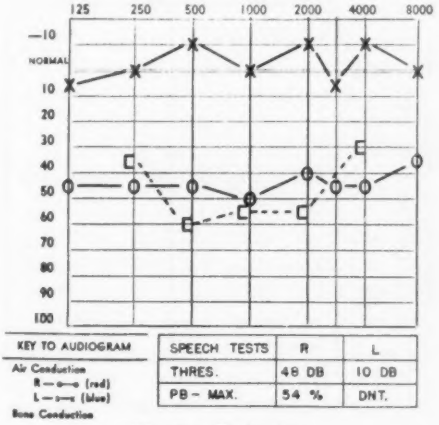

Fig. 2a.—Patient 2.

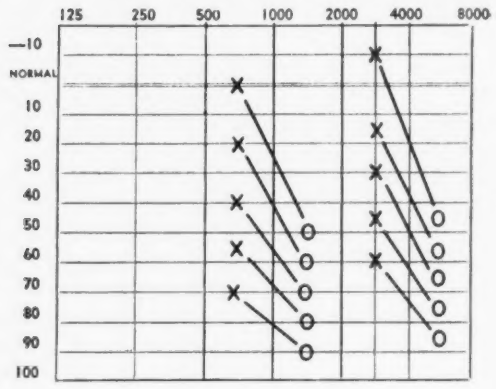

Fig. 2b.—Patient 2.

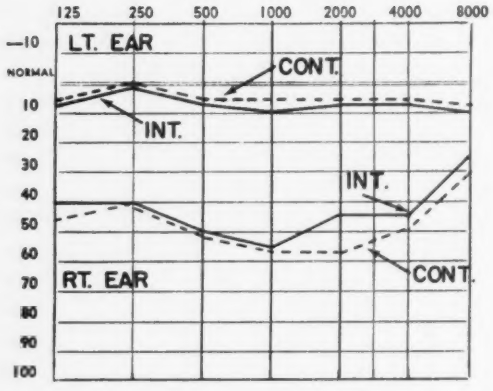

Fig. 2c.—Patient 2

Figure 2d shows three-minute threshold tracings for fixed frequencies in the patient with Ménière's disease. In contrast to Figure 1d, the continuous tone threshold remains quite stable over time. Indeed, the continuous and interrupted tracings are virtually indistinguishable.

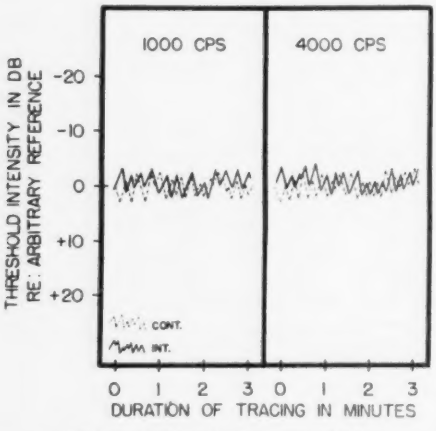

Fig. 2d.—Patient 2. Test ear: right.

According to our experience to date, this finding appears to be uniformly present in end-organ lesion. The threshold tracings of continuous fixed frequencies over time have all been quite stable. Figure 3, for example, summarizes tracings obtained in eight cases of sensorineural loss, in whom the clinical history, diagnosis, and presence of loudness recruitment led to the presumption that the loss was of cochlear origin. Recruitment was established by monaural, bifrequency loudness balance (Reger method) in five cases and by alternate binaural loudness balance (Fowler method) in the other three cases. The open circles in Figure 3 represent the mean interrupted-tone

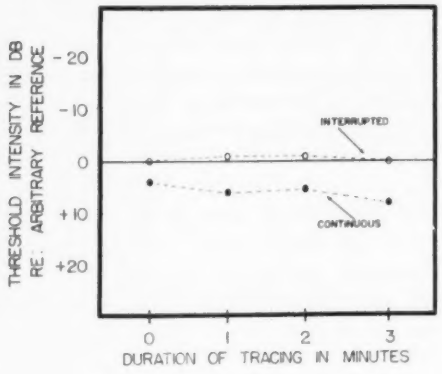

Figure 3

threshold tracings. Six of the eight tracings were done at 4000 cps; the remaining two at 1000 cps. Although there is an initial intensity discrepancy between the two types of thresholds of about 5 db., the mean tracing for the continuous tone drops only an additional 3 db. over a three-minute period.

We certainly do not interpret these results to mean that continuous and interrupted tone thresholds are identical in cochlear losses. The slightly poorer threshold for the continuous tone, exemplified by the fixed frequency tracings in Figure 3, appears to be fairly general at high frequencies in ears with recruitment. Our results do not deny the reality of Dix and Hood's observations in cochlear pathology, but they indicate that, under carefully controlled conditions, the magnitude of these effects is relatively slight. In contrast, when we do observe these effects in an extreme degree, we find them in cases of eighth-nerve rather than cochlear lesion.

Comment

The findings presented above are, as yet, based on an extremely limited number of observations, particularly in connection with eighth-nerve losses. In spite of this limitation, however, we believe it important to report these results because of what we feel to be their significant implications for both auditory theory and clinical practice.

The fact that confirmed cases of damage to the eighth nerve have exhibited extreme adaptation challenges one to speculate regarding the underlying mechanism. This speculation must be tentative, but it is worthy of mention because of its unique implications, provided subsequent observation and analysis confirm it.

In this connection, consider the details of these phenomena as they were manifested by the patient whose responses are summarized in Figures 1a through 1d. The following facts are the pertinent ones.

First, surgery performed subsequent to these tests revealed a large tumor in the left cerebellopontine angle. This tumor extended into the internal auditory meatus. Thus, the site of major pathology was along the trunk of the eighth nerve.

Second, the lesion had induced sufficient destruction of auditory function so that a substantial hearing loss was encountered, irrespective of the manner of testing. Presumably, some destruction of neural elements (and possible secondary degeneration of associated cochlear structures) had taken place.

Third, the lesion had not eliminated auditory responses entirely. Some neural units were still excitable, and the function of the organ of Corti was sufficiently normal to activate them.

Fourth, the lesion had induced an abnormality of such nature that somewhere within the sensorineural system sustained response could not be maintained. Thus, if either a continuous stimulus of fixed frequency or one of varying frequency was employed, the ability to respond at threshold deteriorated appreciably. Conversely, periodic interruption of the threshold-tracing stimulus eliminated any manifestation of excessive adaptation. For example, a continuous tone of 1000 cps (Fig. 1d) completely obliterated the patient's auditory response within half a minute after onset. However, no change in threshold intensity was evident throughout a full three minutes when the stimulus was interrupted three times per second.

Fifth, and this fact is particularly critical, the statements just made regarding hyperadaptation under continuous stimulation are not applicable for frequencies below about 170 cps. As mentioned earlier, and as schematically shown in Figure 1c, the Békésy tracings for the interrupted and the continuous tones are indistinguishable below this frequency. In contrast, sharp adaptation to continuous stimulation is evident by the time the frequency has reached 250 cps.

The full implication of these results becomes apparent when one recalls that the volley phenomenon characterizes neural response at low frequencies[5,6]; that is, the neural units responding to the cochlear microphonics tend to be activated once for every cycle in the stimulus. The consequence is that a 250-cycle tone, for example, initiates twice as many volleys as a 125-cycle tone. It likewise gives the neural units only half as long to recover between excitations.

This difference is not critical in a normal auditory system, where the absolute refractory period is probably as brief as about one millisecond and where sustained responses at rates between 250 and 450 impulses per second may be inferred from observations made on animal preparations.[8] However, this difference seemed to be critical in the case we are discussing. One is encouraged to propose the hypothesis that here continuous stimulation at 250 cps induces prolongation of the refractory periods of those neurons activated by the stimulus. Under such circumstances of delayed recovery, too rapid a train of sound waves, appropriately transformed to a pattern of cochlear microphonics, might maintain desensitization of the individual neurons. It could be that this desensitization is highly localized and is limited to the site, along the eighth nerve, where the tumor is found. Again, it might be a more diffuse disturbance in function resulting from a more generalized disruption in the physiology of the neurons involved. In either event, the end-result would be inhibition of responses. A sufficiently leisurely train of waves, i. e., 125 cps, could allow enough recovery of function for transmission of the signal. The same could be true when short intervals of rest are allowed, as occurs when the stimulus is interrupted in the manner previously described. Thus, we may have here a practical example, in aural pathology, of the all-or-none law of neural behavior: namely, essentially unchanged response, insofar as one can judge from the end-result, until the condition is reached where transmission of impulses is obliterated.

At the clinical level, we urge that threshold tracings for continuous tones of fixed frequency over time be obtained in all cases of suspected eighth-nerve lesion, because we believe that this may ultimately be one of the few, or perhaps, the only auditory symptom that can reliably differentiate cochlear from eighth-nerve lesion. Goodman,[9] for example, has recently demonstrated, rather convincingly, that neither loudness recruitment nor speech discrimination can serve as an infallible differentiating sign. In his series of 18 cases with confirmed retrocochlear lesion, recruitment ranged from complete absence to complete presence, and discrimination loss ranged from none to total.

Such findings are discouraging to the clinician. They emphasize the urgent need for a full-scale frontal assault on the important problem of discovering a set of auditory symptoms unique to eighth-nerve lesions. Our purpose in presenting the above results is to suggest that response to sustained stimulation over time may hold promise as a fruitful avenue of attack.

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A Comparison of Monaural and Binaural Hearing Aids for Listening to Speech

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With the recent development and distribution of hearing aids incorporated into eyeglasses, there has come an automatic reawakening of interest in providing binaural sound amplification for hard of hearing persons. Although the effort to achieve a practical binaural hearing aid has been long and persistent, it is only in the eyeglass models that many disadvantages and problems seem to have been minimized or eradicated.

Mere logical reasoning seems basis enough for the thought that binaural hearing is more satisfactory than monaural; and if one were to give total credence to the claims and inferences of certain hearing aid manufacturers, it would seem that every person suffering from a binaural hearing loss should profit from sound amplification received in both ears. In recent months rather impressive testimonials have been appearing in trade journals supported by hearing aid manufacturers and dealers.[1,2,10,13] Among other things, binaural amplification is reputed to achieve the following: "Speech intelligibility is greatly improved. . . . With the use of binaural hearing you have placed into action the full power of both ears."[10] Bergman has stated that "two-eared, or binaural, hearing allows us to make fine, extra judgments about sound and to listen selectively to one of several sounds." He has said that the advantages of binaural hearing include (1) localization, (2) discriminative selection, (3) speech sound discrimination, (4) ease of listening, (5) sound identification, and (6) improved sound fidelity. And he has offered the opinion that the achievement of these hearing goals may be opened up by the use of the binaural hearing aids.

The whole effect of speech intelligibility under sound amplification by hearing aids has been questioned by Myklebust. At the same time, he apparently thought that a binaural type of aid might alleviate certain difficulties in speech perception:

> Through a hearing aid the fundamental process of separating world of sound into foreground, background, important and unimportant is grossly disturbed. The way in which hearing aids affect auditory perception is urgently in need of scientific study. Perhaps the new hearing aid equipment using two microphones, and which makes depth perception (foreground-background) possible, will bring untold benefits to the hearing impaired during the next few years.

One of the well-known and long-time advocates of binaural hearing aid fittings is Day, who has written from personal experience with a quasibinaural fitting:

> I discovered that by wiring my hearing aid with a receiver for each ear I could understand much more than I could with a receiver in only one ear. This procedure, however, has its drawbacks. With both ears plugged by receivers it is very difficult to modulate one's own voice. It took nearly two years before I could control the modulation of my voice satisfactorily. This is probably the sole reason that so few individuals will wear a hearing aid with receivers in both ears, though I consider it a small price to pay for better hearing.

Carson seems to have taken issue with Day's suggestion as to the singleness of reasons why hearing aids generally and binaural aids specifically may not provide for all needs of the acoustically handicapped individual. She stated:

> Hearing aid users, using only one ear have the same problem [as the person with unilateral deaf-

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ness], but their psychologic attitude is somewhat different. Neither the hearing-aid user nor the patient with a loss in one ear has the ability to localize sounds. If spoken to on the side of the deafened ear, he may miss what is said completely or hear it imperfectly, depending on the physical acoustical situation and the degree of his hearing loss. . . . Unilateral hearing loss is a definite handicap and should not be minimized.

A hearing aid is not the answer to this problem for bilateral hearing cannot be restored in this way. . . . Even if there is enough residual hearing in the deafened ear to use a hearing aid it is impossible to balance a normal hearing ear and an unaided ear as is done for eyes by glasses. The patient will not tolerate the hearing aid but will resort to using his one good ear, adjusting his position as best he can.

Whether one would be justified in projecting Carson's statements to a situation in which a binaural hearing aid seeks to improve two disparate hearing losses is a moot point.

Doerfler has been quoted as favoring binaural use of hearing aids and as believing that discrimination of speech over background noise is one of the benefits.[1]

Binaural summation has been subjected to rather careful study by investigators over a period of many years. Hirsh [6] pointed out: "That a supraliminal auditory stimulus sounds louder to two ears than to only one has been known since Seebeck made his observations a little over a century ago." Contemporary studies in this area have left no apparent doubt that, under controlled conditions, the phenomenon of binaural summation does obtain.[3,12,14] However, it should be pointed out that binaural summation appears to be dependent, in part at least, upon the two ears being equated for their threshold levels. The presence of background noise also appears to disrupt the summative characteristics of the ears. Hirsh [7] stated: "For listening to at least certain stimuli in the presence of loud thermal noise, two ears are not better than one."

Keys investigated binaural and monaural hearing with groups of normal hearing and hard of hearing persons. Among other findings he pointed out that "the gain from binaural hearing decreases as the frequency increases until at 8000 cycles per second the binaural threshold and the better ear threshold are approximately the same." As far as the application of his findings to hearing aids is concerned, he believed that benefits would be derived only if two transmitters and two receivers were used, each set being adjusted to the individual ear.

Larr and Webb conducted a study in which localization of sound was investigated binaurally and monaurally. They reported that 25 of 30 examinees located the source of sound consistently better by means of binaural hearing. Subjective impressions also were reported to favor binaural hearing.

From available research data coupled with reason an impressive case could be built in favor of binaural hearing aids. At the same time it must be recognized that much of the information gathered to date on the summative properties of binaural hearing has been obtained from subjects chosen because of their normal hearing. Further, there is considerable empirical evidence to demonstrate that ears presenting a loss in acuity often do not respond to sound amplification in the same manner as normal ears. And also, one is faced with the fact that not all hard of hearing persons benefit from a hearing aid, even of the monaural type. These considerations seem to justify a careful study of binaural hearing aids and their sound-amplifying properties in a period when their merits are being proclaimed enthusiastically by manufacturers and dealers.

Problem

The present study undertakes to investigate one facet of binaural hearing aid performance. It tests the hypothesis that binaural sound amplification, as provided by commercially built hearing aids, results in better speech comprehension by hard of hearing persons than monaural amplification similarly produced.

Experimental Subjects

The subjects for this study were hard of hearing patients at the Mayo Clinic who were referred

for evaluation of hearing aids. From the outset it was recognized that not all of the acoustically handicapped patients seen would accommodate to the structure of the study. Therefore general criteria for selection were established and followed:

1. No specific age limits were set; however, in each case it was determined that the patient could understand and follow test procedures.

2. No discrimination was made in terms of the sex of the prospective subject.

3. Each subject chosen had sufficient hearing in each ear to respond to the test instruments.

4. The type of hearing loss was not used as a basis for selection.

5. Previous hearing aid experience was not a requisite for participation.

6. All subjects were required to have a sufficient knowledge of English so that lack of comprehension of test words would not be an uncontrolled variable.

Thirty persons eventually were included in the experimental group. Of these, 17 were men and 13 women. Their ages ranged from 28 through 80 years, with the mean at 58.5 years. Nine of the subjects had had some previous experience with a hearing aid, varying from sporadic use to 15 years of consistent use. Mean better ear loss was 40.7 db. as measured by speech reception thresholds, while mean poorer ear loss was 48.5 db.

Equipment

All test procedures were conducted in a sound-treated room in the Section of Otolaryngology and Rhinology of the Mayo Clinic. Audiometric equipment normally available was used for all of the evaluations made. The apparatus included a Maico E-2 clinical pure-tone audiometer, an Altec-Lansing speaker, a Magnecorder Type PT 5-Ah tape recorder and playback, and a Rek-O-Kut Model T-12 playback unit, all serving as partial components of a custom-built audiometric system. Test signals could be introduced under carefully calibrated and uniform conditions. A separate talk-back system allowed patient response to be monitored in the adjacent control room. Recordings of the Harvard PB word lists and the P. A. L. Auditory Test No. 14 spondaic words were used as speech signals. A commercially available hearing aid of the binaural eyeglass type was used throughout the experimental period. A wide variety of standard plastic ear inserts was available to assure a uniformly tight fit in the ear canal of a given patient, and thus prevent auditory feedback.

Test Procedure

Prior to measuring the benefits with the hearing aids, each patient was given a battery of audiometric tests to establish the nature and extent of the individual hearing loss. For purposes of test uniformity a standard routine was followed, and the entire sequence of tests with a given subject was performed by the same examiner. Both experimenters had become thoroughly familiar with all phases of the test sequence before collection of data was initiated. The prehearing testing included determinations of pure-tone air-conduction and bone-conduction threshold, and of monaural speech-reception thresholds using P. A. L. No. 14 spondaic words; determinations of monaural levels of most comfortable loudness using live, connected speech; determinations of monaural thresholds of discomfort, and tests for monaural speech comprehension using Harvard PB words, and for a free-field speech-reception threshold using the P. A. L. No. 14 test.

On the basis of this routine audiometry the ear to be fitted with the monaural hearing aid was selected, by generally recognized considerations. In cases where the hearing was essentially the same for each ear, patient preference coupled with the results of speech comprehension scores led to a decision as to the ear to be tested.

Prior to each test sequence the eyeglass hearing aids were carefully inspected to be certain that both units were in correct operating condition.

In order to control the variable of patient fatigue which might affect test results, the sequence of monaural-binaural testing was rotated from subject to subject. This procedure resulted in 15 of the subjects' receiving the monaural test series first, while the other 15 received the binaural tests first.

As was indicated earlier, suitable ear inserts for the individual patient were selected from an assortment available which assured a sufficiently tight fit in the ear to prevent auditory feedback even with a high gain setting of the hearing aid.

If the monaural fitting was to be tested first, the hearing aid glasses were placed in position and the ear mold, properly attached to the hearing aid, placed in the selected ear. The hearing aid for the selected ear was then turned on, and with the active assistance of one of the experimenters the volume was adjusted to a level subjectively comfortable to the patient. The sound source used uniformly for this procedure was connected speech at a conversational level by the experimenter at a distance of 6 ft. from the patient. Especially in the case of subjects with no previous hearing aid experience, encouragement was given to take sufficient gain to provide a distinct difference between aided and unaided conditions.

With the hearing aid now operative, the patient's monaural free field aided speech-reception threshold was measured in a manner similar to that described above. After this, speech comprehension was tested with recorded phonetically balanced words

TABLE 1.—*Comparisons of Monaural-Binaural Performance*

Comparison	Subjects	Mean Speech-Reception Thresholds (P. A. L. No. 14 Test)			Mean Speech-Comprehension Scores (Harvard PB Words)		
		Monaural	Binaural	Diff., Db.	Monaural	Binaural	Diff., %
Hearing aid experience							
Previous	9	30.4	29.0	1.4	37.1	42.0	4.9
None	21	21.0	18.9	2.1	59.4	60.9	1.5
Severity of hearing loss							
Mild	7	16.9	15.1	1.8	65.1	65.1	0.0
Moderate	16	23.4	20.5	2.9	53.9	57.1	3.2
Moderate-severe	7	31.7	31.7	0.0	37.7	40.9	3.2
Subjective reaction							
Positive	10	24.6	19.4	5.2	55.2	61.6	6.4
Negative	15	22.7	21.7	1.0	52.0	50.7	—1.3
Unaided disparity between ears							
Narrow (0-9 db.)	24	23.1	20.7	2.4	52.6	55.1	2.5
Wide (20+ db.)							
Without No. 18	5	26.4	28.4	—2.0	56.0	53.6	—2.4
With No. 18	6	26.7	26.7	0.0	53.3	55.7	2.4
Total group	30	23.8	21.9	1.9	52.7	55.2	2.5

given at a conversational level of loudness. Patient response to stimulus words was monitored by an examiner over the talk-back system, the process being facilitated by the fact that the subject was constantly under the examiner's direct view. No difficulty was experienced in evaluating the patient's repetition of test words. Data from the testing process were tabulated on previously prepared forms adapted from standard Mayo Clinic audiometric record sheets.

Upon completion of monaural testing, the patient was fitted with the binaural components of the eyeglass hearing aid. Acting with the assistance of the examiner, the patient adjusted the volume of each hearing aid separately until he was aware of hearing through each instrument and was satisfied that the over-all amplification was comfortable for perceiving speech at a conversational level. Once these adjustments were made, the testing sequence was repeated. Test lists of words were varied so that learning through repetition would not influence the performance of subjects.

At the conclusion of the testing sequence the patient was asked about his subjective reaction to binaural and monaural sound amplification. Questions were phrased in a manner which would force the subject to make his own decision as to his preference, if any.

Results

Data collected on the basis of the test procedure described were studied from several different approaches. The results of these comparisons appear in Table 1. It is to be noted that no tests for statistical significance were applied to these data because in many cases it became obvious under inspection of data that little real difference obtained between means being compared. Further, the limited numbers of cases involved in many groups being evaluated precluded the use of even small-sampling techniques in establishing significance of differences between means.

In Table 1 it will be observed that 9 of the 30 subjects had had brief to extensive previous hearing aid experience. Both they and the 21 who had had no previous hearing aid experience benefited slightly from the binaural fitting. Speech comprehension scores for subjects with hearing aid experience indicated that they did somewhat better with the binaural fitting than those with no previous experience.

When the relative severity of hearing loss was considered, it was found that subjects falling into the category of moderate hearing losses tended to achieve greater benefit from the binaural fitting than those with milder or severer hearing losses.

Comparison of subjective reactions to the binaural and monaural fittings in generally positive and negative terms showed that the

TABLE 2.—*Range of Differences Between Binaural and Monaural Speech-Reception Thresholds*

Threshold Differences, Binaural to Monaural, Db.	Subjects	Combined Results	
		No.	%
−10	1	1	3.3
− 8			
− 6			
− 4	2	5	16.7
− 2	3		
0	7	7	23.3
+ 2	7	13	43.3
+ 4	6		
+ 6	1		
+ 8	1	4	13.3
+10	2		

persons who responded favorably to the binaural fitting also achieved better results with that fitting than those who responded negatively. In this regard it should be noted that of the 25 persons from whom this response was received, only 10 favored the binaural fitting.

Table 1 also contains comparisons made on the basis of differences between better ear and poorer ear speech-reception thresholds. Twenty-four of the patients fell into a group having less than 10 db. between thresholds. These persons tended to do better with the binaural fitting in terms of speech-reception thresholds and speech-comprehension tests. Of the remaining six patients, who had a disparity of 20 db. or more between ears, one subject (No. 18) presented results so far removed from the mean of the group as to have a marked influence in all comparisons which were made. The effect is made apparent where the test scores of patients with wide disparity between ears are listed. When No. 18 is excluded, the values from the group are favorable to the monaural fitting of the hearing aid; however, when No. 18 is included, speech-reception scores show no difference, while comprehension is slightly better with the binaural fitting.

Of obvious interest is the manner in which the entire series of 30 subjects responded to the monaural and binaural testings. As a total group, they produced a slight difference in speech-reception threshold and speech comprehension favoring the binaural fitting.

Table 2 displays the range of differences in speech-reception thresholds between monaural and binaural fittings of the eyeglass hearing aid. It will be noted that six persons achieved somewhat lower thresholds with the monaural than with the binaural fitting. Seven persons had no difference between thresholds. On the positive side, 10 persons (33.3%) achieved a reduction of speech-reception threshold difference by amounts of from 4 to 10 db.

From Table 3 it will be seen that 43.3% of the subjects achieved somewhat higher speech-comprehension scores with the monaural fitting than they did with the binaural fitting. However, 53.3% comprehended speech better with the binaural fitting.

When individual subject performance was analyzed, it was found that 10 patients (33.3%) achieved improvement with the binaural fitting over the monaural in both speech-reception and speech-comprehension measures. Three subjects did more poorly

TABLE 3.—*Range of Differences Between Monaural and Binaural PB Test Scores*

Score Differences, %	Subjects	Combined Results	
		No.	%
−14	1		
−12			
−10			
− 8	2	13	43.3
− 6	2		
− 4	1		
− 2	7		
0	1	1	3.3
− 2	1		
− 4	6	9	30.0
− 6	1		
− 8	1		
−10	4		
−12	1		
−14	1		
−16		6	20.0
−18			
−20			
−22			
−24			
−26	1	1	3.3

with the binaural fitting on the basis of the two measurements.

Comparisons between the results of test subjects presenting conductive and perceptive hearing losses and those presenting perceptive losses would be of value; but since only 5 of the 30 subjects in this series presented losses of mixed or conductive types, the significance of the results would have been questionable.

Summary and Conclusions

Thirty hard of hearing persons were tested for speech comprehension with monaural and binaural fittings of a commercially available hearing aid of the eyeglass type. Tests were administered under standard conditions in the Section of Otolaryngology and Rhinology of the Mayo Clinic. Differential results were not statistically significant; however, there appears to be a trend favoring the binaural over the monaural hearing aid fitting in respect to speech comprehension in quiet surroundings. Whether a binaural hearing aid enhances ability to localize sound and what effect noisy environments impose upon relative test performances were potentially significant factors this study did not attempt to investigate.

From the trends taken by the findings of this study, it seems that the binaural fitting of a hearing aid should be an individual matter, and that demonstrated benefits should be reconciled against such other considerations of initial cost, differences in upkeep, and the relative discomfort of wearing two hearing aids.

Mayo Clinic (Dr. Hedgecock).

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Case Reports

Exsanguinating Hemorrhage Twenty-One Days After Tonsilloadenoidectomy

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Postoperative tonsilloadenoidectomy hemorrhage is quite common, noted usually within the first 24 hours. However, the period from the 5th to the 11th day is the time when late secondary hemorrhages are most apt to occur. The case reported here followed the usual pattern of having the hemorrhage at intervals of from six to eight days. The final hemorrhage on the 21st day ended in death.

The patient, a 4-year-old boy, entered the hospital March 18, 1955, at 8 a. m. His father, mother, sister, and three brothers were all living and well. In March, 1954, the patient had measles. A recent chest x-ray was negative. A mouth breather, he had frequent sore throats and colds with a cough. For the three days prior to surgery he had been given procaine penicillin and vitamin K orally three times a day.

Physical findings were chiefly normal; temperature was 98.6 F orally, pulse 72, respiration 20. Eyes and ears normal. Nose congested, uvula elongated, adenoids enlarged, tonsils cryptic. Lungs clear; no rales were heard. Heart was regular; no murmurs were noted. Abdomen was soft and scaphoid; no hernia present. Tentative diagnosis: enlarged adenoids and enlarged cryptic tonsils.

Laboratory findings were as follows: urinalysis—color, straw; character, clear; pH, 6.5; specific gravity, 1.014; albumin, negative; W. B. C., occasional; R. B. C., none; epithelial cells, 0-1; casts, bacteria, and crystals, none. C. B. C. was as follows: R. B. C., 4,100,000; W. B. C., 12,000; Hb. 80%, 12 gm., color index 0.9, clotting time 5½ minutes, bleeding time (Duke) 2½ minutes (normal, 30 seconds to 3 minutes), differential: polymorphonuclears, 43; lymphocytes, 57: eosinophils, 1; stab forms, 3; segmented forms, 40.

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From the Department of Otolaryngology, San Diego County General Hospital, and The Bio-Medical Research Institute.

At the time of admission his temperature was 99.2 F (R). At 11:30 a. m. the patient was given a pentobarbital (Nembutal) suppository, 1 grain (60 mg.). At 12:15 p. m. he was given atropine, 1:300 hypodermically and was taken to surgery. At 1:30 p. m. he was given vinyl ether (Vinethene) and ether for the anesthetic. At 1:39 p. m. the tonsilloadenoidectomy was begun; at 2:10 p. m. surgery ended.

Procedure.—First, the adenoids were resected with the adenotome. The tonsils were removed by dissection and snare. Bleeding was controlled by pressure, and a suture was placed in the left fossa to further control bleeding. At 2:30 p. m. the patient was returned in good condition to his room. His pulse was 130.

Progress.—There, he was given cyanocobalamin (vitamin B_{12}), 100μg. hypodermically, vitamin K, 10 mg. hypodermically, and procaine penicillin, 600,000 units I. M. His temperature at 6:00 p. m. was 99 F (R), pulse 92. He vomited a little, then slept until supper time. At 5:00 p. m. he had ice cream and milk. There was no bleeding, and no subsequent vomiting. After supper he was given a pentobarbital suppository, 1 grain, and he then went back to sleep. On the next day, (March 19, 1955), his temperature at 4 a. m. was 99 F (R). He refused breakfast and liquids. He was discharged at 12:15 p. m., apparently in good condition, and he walked out of the hospital with his mother and brother.

Postoperative Care.—The patient returned to the physician's office on the sixth postoperative day, and all was well. The following day he had a hemorrhage from the left tonsil fossa. Consulted by phone, the doctor suggested bringing the child to the office. The mother did not consider the hemorrhage severe enough to do so. She failed to keep the appointment which he scheduled for the next day.

On the 15th postoperative day, bleeding severer than on the 7th day occurred, and the child was seen in the doctor's office. Although pale and listless, he seemed to be making satisfactory progress. He was given vitamin K by injection.

On the 21st day, a sudden, extremely heavy hemorrhage occurred in the left tonsil fossa. The doctor could not go to the home immediately. The police, who were called, took the child to a nearby hospital. He was dead on arrival. An autopsy made by the coroner revealed a 3 mm. open artery in the left tonsil fossa.

The family filed suit[1] against the physician and at the trial attempted to prove negligence on his part. However, the jury voted 11 to 1 in his favor, and the case was dismissed.

Comment

It is not the purpose of this report to go into a lengthy discussion of post-tonsilloadenectomy hemorrhage or the clotting mechanism of the blood. Recent literature on tonsilloadenoid hemorrhage has been extensively covered by Bellucci,[2] while Coyle[3] has summarized the present-day theories on the clotting mechanism of the blood.

After reviewing the literature and the events in this case, I have come to the conclusion that the period between the 5th and 11th day is the time when the patient must be watched for secondary tonsilloadenoid hemorrhage. The healing membrane usually sloughs out during this period and occasionally unplugs the ends of previously severed arteries, causing bleeding. This can be slight to heavy. The tendency of increased severe bleeding with each episode, as with this case, must be taken as an indication that the involved artery is not producing an adequate end-arterial closure. Therefore, it seems wise when this event occurs for the doctor to take the necessary precautions to avoid an exsanguinating hemorrhage. Some cases require ligation of the involved artery or suturing of the tonsil bed. All cases should receive antibiotics when and if indicated. Studies of the clotting mechanism of the blood must be made and treatment instituted to correct any abnormalities.

Summary

Report is made of a 4-year-old child who developed an exsanguinating hemorrhage 21 days after a tonsilloadenoidectomy. Procedures are outlined which might prevent a similar occurrence.

233 A St. (1)

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Conductive Deafness, Seventh Nerve Paralysis, and Absence of Vestibular Response During Pregnancy

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This is a presentation of unusual findings in pregnancy which is of interest to those concerned with obstetrics, neurology, and otolaryngology.

Report of a Case

During the last trimester of her pregnancy (beginning October, 1956) this 20-year-old white woman noted an immobility of the right side of her upper lip and loss of hearing in her right ear. On Nov. 13, 1956, she was examined by a physician, and a right facial palsy was noted. She had had no history of headaches, head trauma, nausea, vomiting, or balance difficulty.

She was referred to the Neurology Clinic at Walter Reed Army Hospital, where she was found to have a right-sided deafness and right peripheral facial palsy. The remainder of the physical examination was within normal limits. The initial impression was that of a possible acoustic neuroma or a possible Bell's palsy with right ear deafness of unknown etiology. An electroencephalogram on Dec. 3, 1956, was normal. Neurological examination revealed hearing loss on the right and almost complete right facial paralysis but with no other neurological abnormalities. At this same time a skull series report was also normal.

On Dec. 14, 1956, this patient was seen in the Ear, Nose and Throat Clinic, Walter Reed Army Hospital. At this time she stated that she had always suspected that she had some hearing loss in the right ear, but this had definitely become worse. On the day she was examined, the facial paralysis was still pronounced. According to the patient, she was worse when she was fatigued. The conductive deafness on the right side was confirmed by tuning forks and audiogram. The loss by air conduction averaged 52 db. in all frequencies and was normal by bone conduction. Caloric examination with minimal and maximal stimulation with cold water revealed normal response on the left and no response on the right. On this same date, the patient was also seen in the Neurology Clinic, where it was felt that the facial palsy was somewhat improved in that she could partially raise the right corner of her mouth and close her right eye. The fifth nerve had always been intact. Acetazolamide sodium (Diamox) therapy, 250 mg. twice daily, was begun.

On Dec. 28, 1956, she was delivered of a healthy girl by spontaneous delivery. According to the patient, the facial paralysis and deafness disappeared seven days after delivery.

On April 18, 1957 she was again examined in the Ear, Nose, and Throat Clinic, and was found to have no further evidence of seventh nerve paralysis. Hearing was normal and the caloric response was normal and equal bilaterally. This was corroborated by the Neurology Clinic.

Comment

This is a case report of unusual findings occurring during pregnancy. Although the exact mechanisms and specific anatomical areas of involvement are unknown, the coincidence of these conditions with the last trimester of pregnancy (origin and termination) appears to be very definite. A very thorough search of the medical literature covering the past 10 years has failed to reveal any similar case. Faris [1] reported three cases of unilateral facial paralysis which occurred in three primiparous women during the third trimester. In these, recovery was also complete within one to two months after delivery. He believed the cause to be a deficiency of vitamins B_1 and E; yet there was no improvement when the intake of these vitamins was increased. In his cases, as was also true in ours, there

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were no symptoms of neuritis (numbness and tingling) or toxicity.

In a review of studies [2,3] concerned with ascertaining the amount of body fluid increase during the last trimester of pregnancy and the loss during the postpartum period, it would appear that this, too, may be an important point of consideration. The increase in total body fluids during pregnancy is more marked during the last three months, and the highest level does not subside until the 8th to 13th day post partum. This increase is primarily in the extravascular compartment, beginning approximately 40 days before delivery. It is felt, since the findings in this patient coincide so closely with this period of increased fluid content and loss, that probably a mechanism of edema or localized swelling occurred which caused these unusual conditions. The exact site of involvement would be difficult to determine. Since function returned, it must be assumed that the function of the seventh nerve, the conductive mechanism of hearing, and the sensitivity of the vestibular apparatus were impaired but not permanently damaged. The facial nerve pressure site is unknown and it would be difficult to localize it in relation to the geniculate ganglion, even though taste remained intact. Since edema or swelling is more apt to involve the large nerves (especially motor) rather than the small ones (especially sensory), the pressure could have been above or below the geniculate ganglion. The deafness may have been due to some disturbance of the ossicular chain or to involvement of the middle ear, but a serous otitis was never evident. Hearing by bone conduction remained normal. The absent vestibular response might be explained by a slowly progressive swelling within the labyrinth or one involving the vestibular portion of the eighth nerve.

A herpetic basis,[4] the Ramsay Hunt syndrome, may be considered. This, in some cases, causes a facial paralysis, perceptive deafness, and the absence of vestibular response. In the absence of pain or herpetic lesions, however, this syndrome is not thought to be a basis for this diagnosis.

It is felt this case is of instructive significance and value for several reasons. It adds another diagnostic possibility to the more commonly suspected and frequently seen conditions when the physician is dealing with facial paralysis, deafness (unilateral), and disturbed vestibular function in a pregnant patient. The prognosis of this case and the three reported by Faris was excellent, and surgical corrective procedures, such as fascial slings and nerve grafts, should therefore be delayed until after delivery. Unless a definite eighth nerve tumor can be demonstrated with certainty, craniotomy should not be undertaken. Treatment thus far has been of little or no benefit in the cases reported.

Summary and Conclusion

Despite the absence of the usual signs of excessive edema or toxemia of pregnancy in an otherwise healthy young primiparous patient, the etiology of this patient's difficulties, from the information now available, could be on the basis of focal swelling and pressure involving the facial nerve, the labyrinth, and probably the middle ear.

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Carcinoma of the Tip of the Nose

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This unusual and interesting case is presented because of a number of very striking and intriguing problems.

A 66-year-old woman was first seen at the Union Health Clinic on Nov. 6, 1957, because of a painful swelling of her left nostril. She gave a history that three weeks previously she developed an irritation in her left nostril, which she picked. She continued to pick her nose until a crust formed, which progressed to a small "pimple" with a yellow head. The tip of the nose began to swell and become extremely tender and painful. When the patient was seen at the clinic, the tip of the nose was acutely inflamed and presented a moderate swelling in the left nostril at the mesial and superior junction of the ala and the septum. The appearance was that of a typical furuncle. She was placed on antibiotics (crystalline procaine penicillin G [Crysticillin], 600,000 units) daily, advised to apply hot compresses and to return in two days. On her return, the swelling had increased in size, was extremely painful, soft to the touch, and fluctuating. Under local (ethyl chloride) anesthesia, an incision was made into the mass. The scalpel entered a cystic cavity, and instead of pus there appeared a profuse flow of dark black blood. The inner wall of the cavity had the appearance of a sloughed wall of an abscess cavity. The antibiotics and hot compresses were continued. However, instead of receding, in the following few days the process increased in size and became so painful that it was decided to hospitalize the patient.

On Nov. 12, 1957, she was admitted to the Graduate Hospital. The patient presented a large swelling of the left nostril which seemed to originate at the mucocutaneous juncture of the septum. It extended through the soft tissue of the columella and septum to the right nostril and began to protrude into the right nasal cavity. It was red on its upper surface, but the lower dependent portion of the growth was black, resembling gangrene in appearance. The skin over the tip of the nose was not involved in this process, but presented an acute red appearance which extended up to the lower border of the bony nasal pyramid. The mass was extremely tender and painful to touch. The temperature and pulse rate were only slightly elevated. The laboratory findings were not significant: hemoglobin 9.9 gm., RBC 3,550,000, WBC 5000; polymorphonuclears 72%, lymphocytes 22%, eosinophils 1%, and monocytes 5%. The urinalysis was negative. The sedimentation rate was 38 mm. in one hour. The hematocrit reading was 34. The blood chemistry was within normal limits.

The process presented such a bizarre appearance that a number of diagnoses were made, such as rhinophyma, infectious cystic granuloma, and others.

The patient was placed on intensive antibiotic therapy—penicillin, sulfisoxazole (Gantrisin), streptomycin, tetracycline (Achromycin), etc.—and local applications of aluminum acetate solution (Burow's solution).

The past history of the patient revealed that she had a thyroidectomy in 1931, an appendectomy in 1932 with an application of radium (purpose undetermined). For the last 20 years she was under continuous medical treatment for "stomach trouble" (repeated studies failed to reveal any organic pathology). In August, 1956, she was hospitalized for diarrhea, but the x-ray studies were negative. In

Fig. 1.—Front view of the tumor showing its origin in the left nostril and extension to the right.

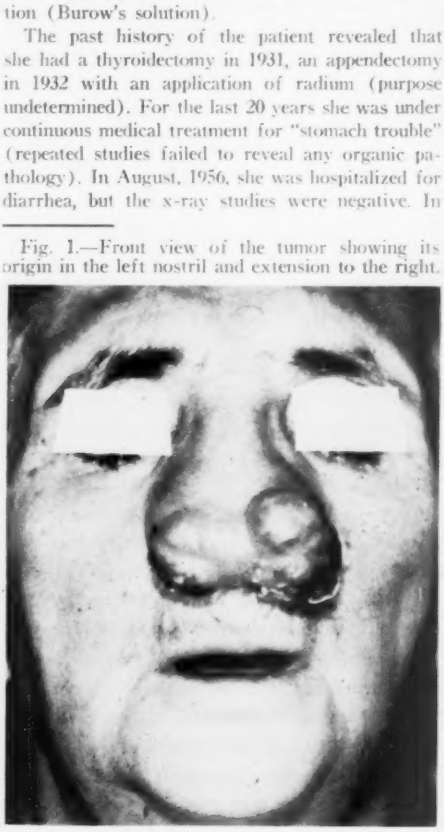

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Fig. 2a.—Left lateral view.

Fig. 2b.—Right lateral view.

April, 1957, she was again hospitalized for epigastric discomfort, nausea, and vomiting. While the routine studies were again essentially negative, a dilation and curettage was performed; the pathologic report of the uterine scrapings was adenocarcinoma of the uterus, for which she received radium therapy.

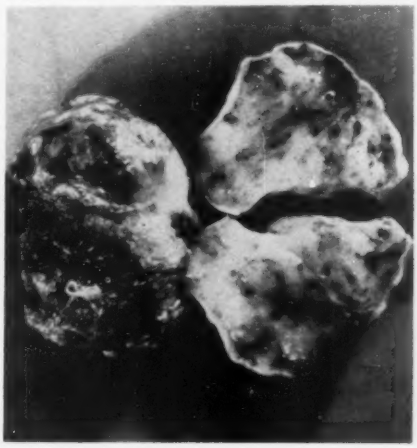
In spite of the intensive therapy, the mass in her nose continued to grow with amazing rapidity, almost doubling in size from day to day. Because of the most bizarre appearance, the patient was presented to a number of clinical conferences that were held at the hospital. At one of these, it was felt that we were dealing with a metastatic carcinoma from the uterus. The growth did not extend backward on the septum or laterally to the lateral wall of the nose or into the sinuses. An x-ray of the sinuses was negative. In view of the inflammatory appearance, a short course of x-ray therapy was given. Figures 1 and 2 present front and lateral views of the patient and the mass.

On Nov. 18, 1957, an incision was made into the mass bilaterally. Again the scalpel entered cystic cavities, and only a collection of dark black blood was released. The cavity walls did not collapse, nor did the growth show any evidence of regression.

On Nov. 20, 1957, sections of the growth were removed for biopsy and culture. The pathologic report was anaplastic carcinoma, adenocystic carcinoma.

On Nov. 26, 1957, under general anesthesia, a transcolumellar incision was made, separating the columella from the upper lip. As this was freed from the septum and elevated, the entire growth could be freed. Laterally, the entire mass was well encapsulated and seemed to be localized to the mucocutaneous junction. There was no extension to either the mucous membrane or the cartilage of the septum, or to the lateral walls of the nose. The mass was removed in toto, but it was possible to preserve the columella and the skin of the tip of the nose. A small section of the alar skin had to be sacrificed. The columella was reattached to the upper lip, and the denuded areas of the alae were covered over by skin. The cosmetic appear-

Fig. 3.—Gross appearance of the tumor mass.



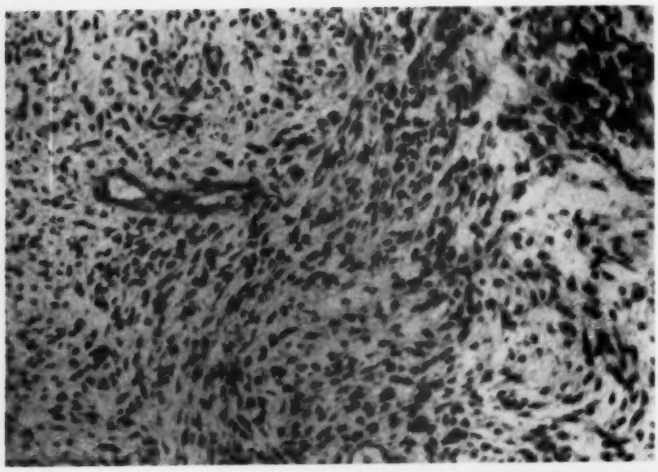


Fig. 4.—Photomicrograph of the tumor of the nose showing the spindle-cellular areas. This pattern could readily pass for a soft-tissue sarcoma, but it actually represents an anaplastic carcinoma.

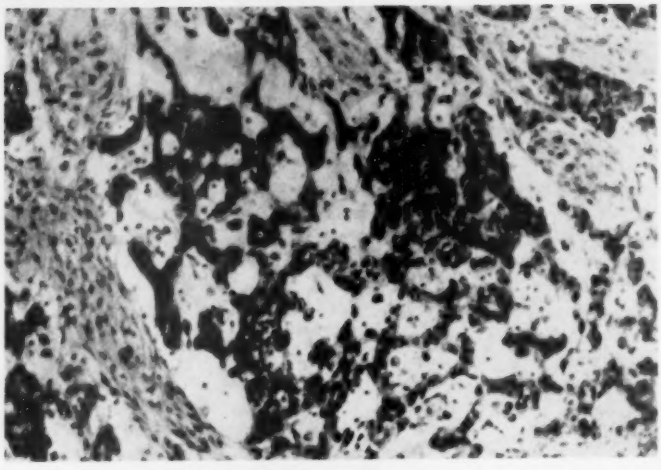


Fig. 5.—Photomicrograph of the tumor of the nose. Here the epithelial component shows cystic or coarsely glandular spaces. Such areas establish the epithelial or possibly the glandular origin of the tumor.

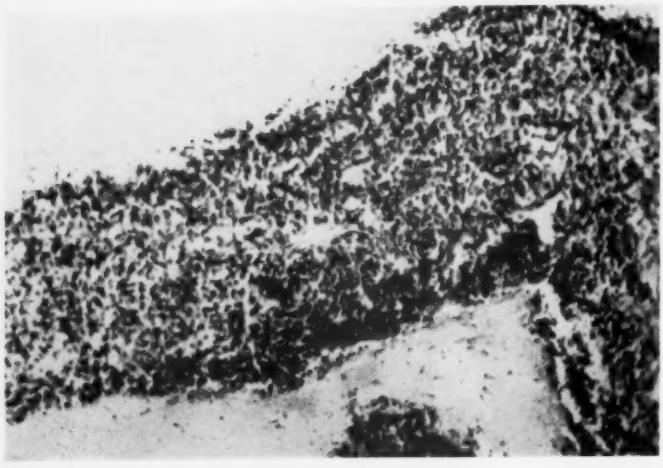


Fig. 6.—Photomicrograph of the cervical tissue. The glandular differentiation is present but very imperfect. The marked anaplasia is evident and makes strict classification unreliable.

ance was rather good. Her immediate postoperative course was rather uneventful. The wound healed slowly with a moderate amount of crusting.

The pathologic findings are as bizarre as the gross appearance of the mass. "The gross appearance presents a number of small cysts beneath the epithelium filled with hemorrhage and the underlying tumor tissue is tense edematous, pink-white, with pin-point cysts filled with coagulum (Fig. 3). Microscopic: in all sections we see a very anaplastic carcinoma that varies from a scattering of atypical cells to fairly regular cords with distinctly epithelial appearance. There is no true glandular formation. In places the tumor becomes spindle cellular. There is no clue from this appearance as to the origin of the growth from a histologic standpoint." (Dr. A. Valdes-Dapena) (Figs. 4 and 5.) A clear-cut diagnosis of the type of tumor was not possible. It was suggested that it might be hemangiopericytoma. On this basis, the slides were submitted to Dr. Arthur Purdy Stout of the Presbyterian Hospital (New York). His report is as follows: "I have studied the sections of the nose tumor with astonishment. I have never seen a tumor like it primary in the skin. It has some of the characteristics of a carcinosarcoma. . . . If this woman had not had a cervical carcinoma, I would have suspected that this case was a malignant mixed tumor or carcinosarcoma. You tell me that the cervical tumor was adenoid with undifferentiated areas. I wonder if it might not have been a carcinosarcoma of the cervix and this nose lesion a metastasis from it." Dr. Valdes-Dapena's report on the microscopic examination of the cervical tissue is as follows: "The islands of the glands are markedly crowded intervening stroma and some of them are partially disintergrating. The lining of the glands are darkly stained, large nuclei, with a considerable number of mitotic figures. In some areas the cells are packed as a sheet losing the glandular pattern. This is unmistakably a carcinoma, and the tumor cells are surrounded by necrotic debris strengthening our diagnosis of malignancy" (Fig. 6). A review of these two reports seems to indicate that we are dealing with two different lesions, and if that is true, it is difficult to establish the fact whether we are dealing with a primary or a metastatic lesion of the nose.

The patient began to complain of numerous symptoms primarily referred to the gastrointestinal tract and a general weakness. A routine gynecological examination failed to reveal any progress of the cervical lesion. In fact, the pelvis was described as normal. There was a secondary anemia, for which she received a transfusion of 500 cc. of whole blood on Dec. 17 and again on Dec. 18, 1957. Deep x-ray therapy to the nose was instituted on Dec. 24 and discontinued on Dec. 31, during which time she received a total of 3,000 r to the nasal area.

Fig. 7.—The appearance of the nose five days before death.

On Dec. 28, the patient developed mental confusion, lethargy, and a marked slowing of responses. The following day she developed a facial (left) weakness. This was followed by a left hemiplegia and a pleural effusion. These complications were due to a metastasis to the brain and the lungs. She died on Jan. 17, 1958. A necropsy was refused.

It is interesting to note that in spite of the extremely rapid growth of the tumor prior to operation, there was no local recurrence of the growth up to the time of her death (Fig. 7).

Comment

This interesting case presented a number of problems:

1. The onset and site of the lesion—its first appearance that of a simple furuncle; a history of picking her nose, followed by swelling, redness, and pain.

2. The gross appearance.

3. The rapidity of growth. The lesion seemed to double in size from day to day.

4. The limitation to one specific area. There was no extension to the septum beyond the mucocutaneous junction; it did not invade the mucous membrane or the carti-

lage of the septum, nor did it extend laterally to involve the sinuses or turbinates.

5. The cystic character of the lesion. These cysts were filled with dark black blood.

6. The absence of metastasis to the cervical lymph nodes.

7. The history of a recent adenocarcinoma of the cervix of the uterus.

8. The bizarre and confusing pathologic appearance, both on macroscopic and microscopic examination.

9. The problem of determining whether the nasal tumor was primary or metastatic from the uterus.

10. The failure of local postoperative recurrence in spite of its previous rapid growth.

11. Finally, the onset of cerebral and pulmonary metastasis.

1737 Chestnut St.

2534 W. Cheltenham Ave.

Society Transactions

CHICAGO LARYNGOLOGICAL AND OTOLOGICAL SOCIETY

Stanton A. Friedberg, M.D., President

Monthly Meeting, Nov. 11, 1957.

Reminiscences. ALFRED LEWY, M.D.

Malignancies of the Paranasal Sinuses. WILLIAM G. HEMENWAY, M.D. (by invitation), and JOHN R. LINDSAY, M.D.

Sixty-two cases of malignant diseases of the paranasal sinuses were reviewed. Fifty-seven were carcinomas, the majority of which were of the squamous-cell variety. Adenocarcinomas have a better prognosis than squamous-cell carcinomas.

Treatment here during the 25 year period has changed from primary x-ray therapy to primary radical surgery. Supervoltage x-ray therapy has been used for three and one-half years. It is too soon to assess its value in these cases, but early results are disappointing. We have no cures in cases treated by x-ray alone or x-ray followed by radical surgery.

Improved results since 1947 are considered due to earlier diagnosis and to the introduction of radical surgery. Six of ten cases in which surgery was employed primarily are alive and well at least for four years.

X-ray therapy is very effective against sarcomas, plasmacytomas, and reticulum-cell sarcomas of the paranasal sinuses. All of five cases treated with x-ray are alive and well.

Disability after surgery is minimized by suitable prosthetic appliances. All postoperative cases who are free of disease are leading reasonably normal lives.

Eighteen case histories are summarized.

Discussion

DR. THOMAS C. GALLOWAY: It has been good to see the advance in the treatment of these distressing lesions. In 1924 at Cook County Hospital we saw many of them, but cures were few. Then New reported 34% cure with the combined treatment of irradiation and heat destruction, first with a soldering iron, then with electrocoagulation. Ohngren's series was about the same. Larsson and Martensson (Larsson L-G., and Martensson, G.: Carcinoma of the Paranasal Sinuses and the Nasal Cavities, *Acta radiol.* 42:149-172, 1954) in 1954 had raised this to over 40% success with combined treatment.

We are, however, not doing much better in diagnosis. A recent case with fairly obvious symptoms had been seen by four men before the last suspected malignancy. It may be excusable not to diagnose malignancy, but it is inexcusable not to think of it. The early symptoms are those of sinus disease, obstruction, purulent secretion, and pain with hemorrhage added. Pain, if it is severe, persistent, and progressively worse, is most important. Diagnosis requires adequate exploration and biopsy. X-ray evidence of bone destruction is late, but usually is present when the diagnosis is made.

I want to revive interest in diathermy. In 1924, Frank Novak taught us electrocoagulation and in our nearly hopeless cases without adequate irradiation it proved very useful. It is so little understood now that some men talk of it as "fulguration," which is a very superficial, ineffective modality. Coagulating diathermy, combined with irradiation, has seemed the most useful technic as demonstrated by New and Figi, and at Radiumhemmet, by Ohngren and Berven, and the last fine series of Martensson and Larsson in 1954.

Young men now seem fascinated by the speed with which they can work on the maxilla. At the Academy it was reported that the maxilla could be removed in two and one-half minutes. The lesion over the alveolar ridge for which this was done appeared to be a local one which might have been destroyed by electrocoagulation, more easily, safely, and with considerably less mutilation.

These tumors do not follow the anatomic conformation of the maxilla. Most of them go up to the ethmoid area, where scalpel surgery would not be effective. When I use surgical diathermy, seeing the tumor as well as feeling its penetration by a coagulating coarse needle electrode, my own results have been much better.

I commend a restudy of the coagulating-diathermy technique in place of these widespread resections. One can get any kind of exposure he needs and so remove under vision any part or all of the maxilla, septum, or ethmoids if necessary. By a combined Mosher incision with a trapdoor flap over the bridge, I successfully destroyed an adenocarcinoma which had invaded both frontals, both antra, ethmoids, and sphenoids, with external distortion. With ingenuity and care one does not need to sacrifice a large amount of uninvolved bone.

It is true that after diathermy healing may be slow, with considerable cicatrization, but this is

minimal if most of the coagulum is carefully removed. One does not get the inoculation metastases or the unnecessarily widespread destruction that may occur after arbitrary anatomic resection.

Most men who see many of these cases agree that there should be a combination of irradiation and surgery. Martensson and Larsson found their cure rate reduced by 15% without surgery. I see some logic in giving a full course of preoperative irradiation with surgical diathermy in one or two months. This is then well tolerated, with good healing, although limits of the malignancy are not so well defined.

Dr. Francis L. Lederer: I can only rise to challenge some of the remarks of Dr. Galloway. It seems to me that on the one hand he made some statements which are acceptable, and, on the other hand, some others that were quite paradoxical. You do not approach malignancy through a keyhole incision. Wide exposure of malignant sites is essential, certainly this is true in sinus malignancy. Twenty-five years ago this operation was thought to be a mutilative one. Nothing has changed the concept of wide exposure and removal except that we do a better job. We have better methods of anesthesia, blood and blood substitutes, and chemotherapy, and certainly our prosthetic appliances and rehabilitative approaches are superior. I do not believe in plunging coagulation needles into tissue adjoining bone. This modality is so uncertain as to control of depth that it is difficult to measure its effectiveness. Dr. Galloway has stated that sloughing bone takes a long time to come away. This is why we require a better understanding of electrosurgery.

We at the University of Illinois have some disagreement on the subject. There are some members of the department who remove the hard palate in every instance so as to enable them to observe the cavity for possible recurrences in the months ahead. We are not agreed that the palate should come out. We are, however, agreed that not "radical" surgery, but "adequate" surgery, is the treatment of choice for malignancy of the sinuses.

Dr. Irwin D. Horwitz: It is interesting that Dr. Hemenway presented consecutive cases. Dr. New did not present consecutive cases; he presented selected cases. We have had 100 consecutive cases at the University of Illinois. There has been one cure by radiation. All of our patients have combined therapy. They all have adequate surgery and full radiation. Forty per cent is our cure rate. I do not think primary surgical approach or primary irradiation is correct. Dr. Hemenway mentioned six sarcomas. In our 100 cases we have had 6 or 7 that were not epidermoid carcinomas. Ohngren had a tremendous number of those cases. There is a tremendous difference in the cure rate. In Ohngren's book there is a chapter on radiation. We think short-wave diathermy good, but ionizing radiation can be better and safer. At the University of Illinois we have a standardized therapy of taking out the orbit in many cases, followed by a full course of radiation.

Dr. Alfred Lewy: Since we are talking about history, I should like to tell you that diathermy is not new. In the early 1920's, my thesis for entrance into the "Triological Society" was "Diathermy by the Needle Method," in which I reported a number of malignancies and my mortality rate was very high. If I remember rightly, I saved only one out of four cases. Of course, diathermy has made great progress since then.

Dr. Thomas C. Galloway: I do not believe that my friend, Dr. Lederer, is quite fair in implying that I would treat malignancy through small openings into the bone. After these many years he should know that I have always insisted on complete exposure and radical destruction. In a recent case with sublabial and suborbital incisions I removed the anterior antral wall, ascending process, and nasoantral block and had complete exposure of the ethmoid and sphenoid area. I saw the whole antral roof and removed most of it but did not have to remove the malar complex or the posterior alveolar ridge. One must get adequate exposure, but I still think that diathermy provides greater safety, with less hemorrhage, shock, and mutilation.

Dr. John R. Lindsay: In a recent report from the Middlesex Group in England their series was divided into a group of untreated cases, a group of early cases, and a group of late cases. It has been our experience with only one or two exceptions to have had all late cases.

In our opinion, when these tumors show evidence of involving the orbit, the orbit should be exenterated. In several cases we have regretted that we did not do so.

Dr. William G. Hemenway: As I am not familiar with Dr. Galloway's technic, I cannot comment on it. I am sure it is successful in his hands. I do not want to give the idea that we do a disfiguring operation on all these lesions. I believe that if I found a lesion that did not involve the anterior wall, or involved only the maxilla or antral wall, I would vary the procedure accordingly, but I think I had bad influence in my younger years over at the University of Illinois because I have no idea of changing my approach.

Benign and Malignant Tumors of the Larynx (motion picture)

CHICAGO LARYNGOLOGICAL AND OTOLOGICAL SOCIETY

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Oscar Sugar, M.D., President
John F. Madden, M.D., Vice-President
Myer Brown, M.D., Secretary

Joint Meeting, Dec. 2, 1957.

Multicentric Glomus Tumor: Report of a Case.

RICHARD A. BUCKINGHAM, M.D., and (by invitation), LORIS PERRELLI, M.D., and KENJE AIMI, M.D.

Glomus tumors occur at one or more of several sites in the body where normal nonchromaffin paraganglial tissue exists. These places are the aortic body, the carotid body, the jugular foramen, and the glomera jugularia of the middle ear as described by Guild.

Histologically, the glomus tumor, or carotid body tumor, resembles the normal accumulation of nonchromaffin paraganglial tissue. There is no histological evidence of malignancy. However, as is known, these tumors enlarge and will cause death eventually by invading vital structures.

The authors present a case where a small, 3 mm., glomus jugulare tumor was found in the middle ear. It was found to be a completely separate independent lesion. In addition, there was evidence of a large jugular foramen lesion because of the 9th, 10th, 11th, and 12th cranial nerve involvements. The authors were able to prove the identity of the tumor in the middle ear by a biopsy but were unable to make a histological proof of the lesion in the jugular foramen; they assume that it, too, is a glomus jugulare tumor.

They point out that the literature contains many cases where carotid-body-like tumors and glomus jugulare tumors were found concomitantly in the same or different sides in the same patient. They suggest, from the evidence of their own case and the evidence of other cases in the literature, that the glomus tumors may be caused by a common exciting agent which causes an outburst simultaneously of glomus tumors in any one or more of the several sites in the body where this nonchromaffin paraganglial tissue normally exists. The cause, however, is unknown.

They suggest that further cases be observed carefully for the possibility of other evidences of the multicentric origin of the glomus tumor.

Musical Hallucinations as a Psychical Seizure: Focal Epileptic Disorder.

ALEX J. ARIEFF, M.D., and (by invitation) JAMES M. BROOKS, M.D.

Although hallucinations are not uncommon as a psychiatric or neurologic disorder, they are uncommon as a part of psychical seizures and have rarely been reported in the literature. It is for this reason that we are reporting the case of a 90-year-old widow who was well until a few years ago except for mixed nerve deafness, when she began to hear voices and noises—first like men talking; then she began to hear songs. She could name 17 compositions. Between the hearing of these songs, she had free periods. These would come on any time of the day and as she lay down to sleep. Sometimes they would stop on sitting up.

Neurological examination was essentially normal except that she had hypertension (240/120 mm. of mercury). An EEG revealed bilateral anterior temporal slow-wave foci. She improved on 45 mg. (¾ grain) of diphenylhydantoin sodium (Dilantin) three times a day with 45 mg. of phenobarbital at night, and thereafter rarely had a similar attack. She was put on a trial of azocyclonol (Frenquel), 20 mg. three times a day, for a period of approximately 10 days. Her hallucinations returned during this period, however, and she was again placed on diphenylhydantoin and phenobarbital medication.

In Penfield and Jasper's monograph on epilepsy, there are four case reports in which music is a part of the seizure, and at times music was elicited when the brain was stimulated. They feel that psychical hallucinations bear the same relationship to the superior and lateral surface of the temporal lobe by sensory seizures or hallucinations due to sensory area of cortex. Psychical hallucinations may be preceded or followed by other phenomena, such as abdominal sensations, buzzing in ears, flickering light, and a sense of familiarity or fear. It may be followed by automatism and amnesia. These other phenomena are evidence that discharge has passed from one anatomical area or system into another.

In our patient, the hallucination was the only manifestation of this focal seizure.

Discussion

DR. ROBERT HENNER: We had a patient about five years ago at the Illinois Eye and Ear Infirmary who presented musical hallucinations. I remembered from medical school that these are usually toxic episodes. I began to question the patient about any drugs which had been used, and discovered that he had been using Bromo-Seltzer. Bromine studies of his blood were done, and the level was found to be 75 mg. per 100 cc. of bromine; he was taken off the Bromo-Seltzer and made a complete recovery from his musical hallucinations.

DR. JAMES M. BROOKS (by invitation): One rarely runs into a problem such as in Dr. Arieff's case. I think this is the second one I have seen in about seven years.

This woman came in because of symptoms related to hearing. She heard music which she knew wasn't there. An audiogram made in 1954 showed a 30 db. loss at frequency 250, which gradually increased to 80 db. at frequency 8000.

The first case of hallucinations I encountered was in 1952, and this patient was also seen by Dr. Arieff. He had a blast injury which caused a hearing loss of nerve type with a decrease of 55 db. at frequency 4000 and 20 db. at 2000. He heard voices which he didn't realize were not real.

Occasionally we do run into the problem of hallucinations, and I think they are best referred to the neurologist and psychiatrist.

DR. ALEX J. ARIEFF: I wish to thank the discussants of my paper. Hallucination can occur from any type of drug toxicity. Many years ago Bromo-Seltzer not only contained bromine, but also contained Cannabis indicus. These both can be toxic drugs. It is unusual for 75 mg. of bromine per 100 cc. of blood to be toxic; however, not impossible. This is almost the lower limits of normal. Cannabis indicus can cause hallucinations. I would favor that as the cause of the musical hallucinations referred to by Dr. Robert Henner.

The Management of the Dizzy Patient: Contribution of the Neurologist.

ROLAND P. MACKAY, M.D.

The word "dizziness," of course, has no precise meaning. It runs the entire gamut from Wallenberg's syndrome on the left to a dizzy blond on the right. Recently DeWeese classified dizziness into two groups: the systemic, in which there are 30 varieties, and nonsystemic, of which there are 29 varieties, for a total of 59. This is really not a classification, but a list of the various conditions producing a sensation which the patient may choose to call dizziness. A definition seems necessary before we start talking about dizziness. Without it we shall be guilty of much nonsense. The word dizziness, as popularly used, has no meaning, but indicates only a strange feeling of some kind, a perturbation which the patient may refer to his head. A goodly number of cases are the result of increased intracranial pressure, syncope, anemia of certain kinds, possibly hypotension; some patients mean spots before their eyes. Contributing to dizziness may be toxic states which cause this type of disorder. Also intracranial tumors may cause some kind of dizziness, particularly those in the posterior fossa which may cause circulatory changes within the head. Another patient may complain of dizziness when he means the very absence of this sensation. For example, any disease which causes cerebellar dysfunction may give ataxia and a sense of instability which the patient may think is dizziness. Even purely psychological phenomena may be spoken of as dizziness. For example, a patient suddenly looking over the parapet of a tall building may experience neurotic panic, often with hyperventilation, leading to dizziness through a relative alkalosis and complex visceral reactions. People may use the word dizziness for confusion, bewilderment, or turmoil caused by crowds. These are all nonsystematized, indiscriminate symptoms, and to be distinguished from genuine vertigo. By definition, vertigo is an illusion of postural change, a false sense of rotation, or some other motion. Some say that external objects seem to move; others feel that they themselves are moving in space. I believe the distinction has no diagnostic value but is rather a matter of terminological preference.

With this definition we may recognize certain types of dizziness. The first is spontaneous vertigo, that without known cause or motion on the part of the individual. The second is hypersensitivity induced by movement of the body or head. Spontaneous vertigo occurs in patients with aural disease, such as Ménière's syndrome accompanied usually by tinnitus and progressive deafness. Other causes may be otitis, mastoiditis, arteriosclerosis, or other vascular changes in the internal ear. In addition to aural factors, spontaneous vertigo may be due to central neurological diseases, of which three examples may be listed. The first is the Wallenberg syndrome caused by thrombosis of the posterior inferior cerebellar artery. Second, multiple sclerosis, with plaques in the medulla, may cause spontaneous vertigo. Finally, the ictal (epileptic) states may appear as a vertiginous experience. Thus, vertigo may be the aura of an epileptic attack.

Induced vertigo, or hypersensitivity to motion, is common in aural diseases. Prolonged motion is another cause, leading to seasickness or carsickness as examples. This type requires enduring motion for its production and is not brought on by

sudden movement of the head. In addition, some people seem so constituted psychologically as to be subject to seasickness. It is as if a certain psychological preparedness predisposes to seasickness. It is worth noting that this vertigo of motion may not be unpleasant. For example, children may turn themselves for the fun of it and enjoy this unique type of vertigo after rotation. The thrill of riding on a roller coaster involves much more than labyrinthine stimulation.

Another type of vertigo is seen in the very common postconcussion syndrome, which consists of a triad of vertigo on motion, headache, and a peculiar hyperirritability or sensitivity to confusion, to noise, and to vibration. Such a patient is irascible and may explode with wrath. So stereotyped is this triad that to me it acquires the validity of an objective sign, and if not taught, should be so recognized in the courts.

There is another group of patients with vertigo upon adoption of certain positions in space, such as lying on the left or right side and not on the back. These may be due to disease in the posterior fossa, particularly tumors. Vertigo consequent upon certain positions rather than motion suggests a mechanical influence. Perhaps the unique position produces extramedullary pressure and so sets up vestibular reflexes. Another type of vertigo is unique and interesting. This is probably a visual after-image of motion. For instance, if one stands on the rear platform of a rapidly moving train and looks steadily at the receding track, and if the train then stops, one will fancy that the track is now rushing forward. Or if one sits in a moving train watching the landscape rush backward, when the train stops the landscape will then seem to move forward. This, then, perhaps is an optokinetic, or kinesthetic negative after-image.

What shall we do about these types of vertigo? Of course, one must first try to diagnose the type, establish the mode of onset, and the course of symptoms, in order, if possible, to find the cause. One must pay particular attention to associated phenomena, such as tinnitus or deafness, which will be highly important in making the diagnosis. Many other things must be sought, such as anesthesia of the face, loss of corneal reflex, Horner's syndrome on one side, etc. They cannot all be mentioned here. Spontaneous vertigo in Ménière's syndrome, often associated with tinnitus and progressive deafness, is of a paroxysmal nature and lasts a relatively short period of time. Such patients may be quite free of any of the symptoms in the intervals between attacks, which succeed each other in clusters and may then come to an end, leaving the patient free of symptoms for a long time, perhaps for years.

Many theories have been proposed to explain this peculiar group of symptoms. I am sure that there is probably more than one cause if we take all cases into consideration. However, the theory is plausible that hydrops of the labyrinth is responsible, because such patients often respond to a salt-free diet, the administration of potassium chloride, 1 gm. (15 grains) three times a day, and reduction of the fluid intake.

Desensitizing histamine injections have been recommended, but in my own experience are not very successful. In my experience the antihistaminic drugs are almost a total failure in the treatment of the vertigo of Ménière's syndrome. Incidentally, in order to prevent seasickness with antihistaminic drugs one must give them before the trip as a prophylactic measure. If one waits until the patient is seasick, it is too late.

It may be necessary to operate on the ear for some of the aural causes of spontaneous vertigo. Some such causes requiring operation are mastoiditis, cholesteatoma, and other otologic disease.

The Wallenberg syndrome as a cause of spontaneous vertigo deserves special attention. It is due to thrombosis of the posterior inferior cerebellar artery. Clinically it may be extremely confusing when first seen, but once seen, it is always recognized easily. There are many clues; the sudden onset, the overwhelming vertigo, seeming to the patient to turn the bed upside down, the dysphonia, the dysphagia, the Horner's syndrome, and the alternating hemianalgesia, the face being analgesic on the side of the lesion, the rest of the body analgesic on the opposite side, and the nausea and vomiting. In such cases one has little that one can do specifically. Often such patients recover from the acute phase, but some may die of thrombotic involvement of the basilar artery. Those recovering may have the alternating hemianalgesia for years. Before leaving this topic, one must say that not all patients exhibit the full syndrome. Multiple sclerosis may cause spontaneous vertigo but it is a rare cause, and remission is usual after a few days or weeks. Such patients may be helped by any symptomatic measures available. If one suspects an ictal process, one must look for the other concomitant factors such as alteration of consciousness. The EEG, which is so diagnostic in epilepsy, is very often not useful in these cases because the discharging area is presumably not accessible to the electrodes. Probably such cases are best treated with dilantin and phenobarbital. What to do about motion sickness and other types of induced vertigo again is complex, and is determined by the cause of the motion sickness. An important element in the management of such cases is the reassurance of the patient, as a prophylactic measure. He may also be prepared with the antihistaminic drugs.

The postconcussion syndrome is a therapeutic quagmire into which many complications and legal

questions enter. One sometimes can't cure these patients because the lawyer won't let them settle until well, and they can't get well until the legal controversy is over. It is better for these patients to go back to their usual activity as soon as possible. It is much better for them to lead useful lives than to sit at home nursing their symptoms. One may give them mild sedation. Dehydration is of no value in these cases.

Finally, if one has a patient with vertigo arising from one side and not the other, one should look for signs of a tumor in the posterior fossa involving the fifth, eighth, and ninth cranial nerves. Tumors in the cerebellopontine angle may be responsible and even localized chronic inflammatory cysts may be the cause. Operation on such a tumor or cyst may be curative.

Management of the Dizzy Patient: Comments on Diagnosis. JOHN R. LINDSAY, M.D.

The first and main problem in any case of dizziness is the etiologic diagnosis. For the purpose of discussion it is convenient to use some type of classification. This can be an etiologic classification based on localization—for example, disorders of the peripheral ear, central disorders, and a third group in which the localization may not be definite.

The only peripheral disorders which I would like to review are those which have been loosely classed as Ménière's syndrome. Under this heading there are two types of disorder from the standpoint of symptomatology and clinical course. One is the group now known as Ménière's disease, or hydrops of the labyrinth, and the other is the syndrome of sudden deafness, or vertigo.

Ménière's disease, the clinical characteristics of which were accurately described by Ménière nearly 100 years before the discovery by Hallpike and Cairns that a hydrops of the labyrinth existed in these cases, usually produces recurrent and violent dizzy spells. The more constant characteristic, however, is the disturbance of hearing. If a case of vertigo has no auditory disturbances, the diagnosis of Ménière's disease cannot be made. The auditory disturbances are sufficiently characteristic that a diagnosis of Ménière's disease can be occasionally made in the absence of dizziness.

The typical auditory findings are a hearing loss primarily for low tones, for air- and bone-conducted sound, a fluctuation in the degree of loss from time to time, the presence of the recruitment phenomenon, disproportionate loss of ability to discriminate speech at maximum intensities, and lowered threshold of discomfort caused by loud sounds.

As the disease becomes advanced, the fluctuations are lessened and the threshold for high tones tends to be impaired also.

When the disease starts in middle life, it very often occurs in cases in which there already is a bilateral high tone deafness of the presbyacusis type. In approximately 50% of 100 cases reviewed by me several years ago Ménière's disease was found to have been superimposed on a bilateral high tone deafness.

The cause of Ménière's disease is not yet understood, but the information so far obtained points to a change in chemical constitution of the endolymph, consisting possibly of an increase in protein molecules, to account for the increased endolymphatic pressure and consequent hydrops. A disturbance of the secretion of endolymph appears likely.

Ménière's disease is clearly differentiated from the deafness and dizziness of sudden onset. In the latter syndrome there is usually one attack of deafness, tinnitus, and vertigo. The deafness varies in degree but affects high tones primarily in most cases or may even be total and does not fluctuate, although there is frequently spontaneous improvement in the first two or three months afterward.

The dizziness is not severe at, or near, the onset, but recovery is gradual and may require from a few weeks to two or three years.

It is common that the vertigo as it subsides becomes a postural symptom and can be reproduced by appropriate posturing of the patient. This is in direct contrast to the typical dizzy spell in Ménière's disease, in which the dizziness, although severe, usually ends abruptly.

There has been much discussion about the cause of these attacks of sudden deafness and vertigo. Very little is known of the pathology. The terms "hemorrhage of the labyrinth" or "toxic labyrinthitis" are often heard. Hemorrhage into the labyrinth, or a hemorrhagic exudate into the labyrinth, is well known in advanced leukemia and in some other systemic diseases with a hemorrhagic tendency. We have recently observed a case of sudden deafness during bishydroxycoumarin therapy. However, the existence of a hemorrhage into the labyrinth in the absence of a hemorrhagic diathesis is probably rare.

A more probable cause is the occlusion of a part of the blood supply, either venous or arterial. A vascular spasm may be the explanation in some.

This type of labyrinthine disturbance occurs at all ages—particularly from the twenties up. Although it may occur in the presence of advanced cerebral arteriosclerosis, this seems to be a minority group. The attacks are most frequently one in a lifetime.

The effect of occlusion of the venous return flow and of the arterial flow on the auditory and vestibular sense organs has been studied in the guinea pig by Perlman and Kimura in our laboratory.

They have demonstrated the degenerative changes to be produced by this type of vascular lesion. While information as to the pathology in the human is not available, these animal experiments illustrate at least one probable explanation of such attacks.

There is a group of cases of sudden deafness and dizziness in which the etiologic agent is most probably a virus. The most classic examples have occurred in acute contagious diseases, such as mumps and measles, but, in addition, there are several well-known syndromes in which the ear is affected in which the etiology is thought to be a virus. Again there is a scarcity of information about pathology. The only cases in which the pathology has been described are those in which the measles virus has damaged the inner ear. One such case was briefly reported by Dr. Hagens, of this Society, some years ago. A bilateral case was reported by us in 1954.

In our cases the damage to the inner ear was limited to the endolymphatic system, an endolymphatic labyrinthitis. The virus had apparently reached the inner ear by the blood stream and, by causing injury to the stria vascularis, had gained access to the endolymph.

In a case of measles previously reported by Nager, there had apparently been a meningoencephalitis with direct invasion of the perilymphatic spaces of the ear from the meninges. This illustrates the probable route by which the complication of deafness and dizziness may occur in the presence of meningoencephalitis. In addition to these two routes of invasion of the inner ear, there are cases in which a direct extension to the inner ear occurs from the middle ear. This has been observed in cases of nonsuppurative otitis media. Whether this represents a direct extension of a virus to the inner ear or a toxic effect is not definite, but there is clinical evidence that either may occur.

These cases of measles illustrated here give some information as to how a virus may attack the inner ear.

One of the responsibilities of the otologist is the early detection of tumor of the nerve or in the cerebellopontine angle.

In cases of unilateral nerve type deafness with or without vertigo the possibility of tumor must be considered. Most cases of acoustic neurinoma are likely to consult an otologist because of unilateral hearing loss or tinnitus; the otologist may have the best opportunity to make an early diagnosis, thereby improving the prospects for successful surgery.

For this reason the routine use of vestibular tests, especially caloric tests, particularly in cases of unilateral progressive nerve deafness, is advisable, since reduced or absent caloric responses are usually found in early stages of neuroma. X-rays are also important.

The development of tests for speech discrimination in recent years has opened up the possibility for earlier detection of tumors affecting the auditory nerve or central auditory pathways. The observation that lesions of the nerve might reduce the ability to understand speech while the thresholds for pure tones remained undisturbed has been confirmed in several cases by the use of discrimination tests for speech. Further development or refinements of discrimination tests may prove to be of greater diagnostic value.

As stated at the beginning of this discussion, the largest clinical group of dizzy patients are those in whom there are no associated auditory symptoms and no localizing central nervous system signs. In this group there may be one attack of vertigo of short duration, one attack with a prolonged recovery period, one attack followed by postural vertigo for months or even years, or repeated attacks. Also the dizziness may not occur in attacks but be a more or less intermittent symptom.

When confronted with the complaint of dizziness, the first necessity is to find out which body system is the source of the disturbance. For example, the visual, locomotor, and circulatory systems may each be a source for a disturbance that might subjectively be described as vertigo. Syncope, Stokes-Adams syndrome, carotid sinus syndrome have all been confused with a vestibular disturbance. A purely psychic disturbance may give rise to the complaint of dizziness.

The presence or absence of a true vestibular disturbance can usually be determined by a careful history and the routine examination. The presence of a postural type of vertigo requires special questioning to bring out, and, if present, it can be verified by postural tests.

Having established the existence of a true vestibular type of vertigo, one should next be concerned to look for certain systemic conditions, blood dyscrasias, anemia, hypertension, infections, et cetera, and to investigate the central nervous system for evidence of disease such as cysts, early tumor, multiple sclerosis, vascular accidents, etc.

In the absence of definite localizing signs that would indicate the location as well as the probable nature of the lesion, there are three or four groups into which most of these cases fall.

There is a group due to infection, thought to be a toxic manifestation. The dizziness may be mainly a postural symptom or, if severer, may be constant, but is usually of a fairly short duration and is relieved if the infectious source, such as an abscessed tooth or sinus, is removed.

There is a small group who have severe vertigo due to a certain drug.

Vertigo is a frequent complaint following head injuries. It is most often postural in character and can be verified by appropriate postural tests.

There is a large group of cases in which the vertigo is probably due to a vascular occlusion which has been localized to some part of the vestibular system.

There have been no definitive reports of the pathology in this type of case, as far as I have been able to determine, until the case reported by us recently. In this case the cause of the severe attack of vertigo followed by several years of slowly decreasing postural vertigo was found to have been associated with a small vascular lesion involving the vestibular nerve at Scarpa's ganglion with degeneration of the superior part of the vestibular nerve.

The clinical course in some of these cases strongly suggests a vascular occlusion. An acute onset with more or less severe vertigo followed by a long period of gradual recovery in which the receding vertigo may be mainly postural is suggestive of a vascular occlusion. The complaint of unsteadiness in walking or making quick movements often persists longer than any demonstrable nystagmus.

Another group which seems to be clinically differentiated is that in which the symptom of vertigo, mainly postural, appears to be related to hypotension or vasomotor instability.

In these cases of vertigo which develop without definite auditory or other central nervous system signs, the vestibular tests vary. A depression of vestibular responses on one side, or a directional preponderance of the nystagmus is of help in localizing the side of the lesion. Certain abnormal or perverted responses to caloric excitation indicate a central lesion.

Certain characteristics of the postural vertigo and positional nystagmus strongly suggest a central lesion.

Experiments now under progress have shown that a positional nystagmus may be produced by localized lesions to various parts of the central vestibular system. It is also known clinically in the human that some inner ear lesions may produce a postural vertigo and a positional nystagmus.

In the management of any case of vertigo the most important step is the diagnosis of the etiology. The patient is usually apprehensive about some progressive disease, such as brain tumor or multiple sclerosis, or is afraid of an impending stroke. The relief which may be obtained by the use of drugs and avoidance of certain movements and positions is helpful, but the reassurance that can be given the patient on the basis of a careful otological and neurological examination is an important part of the treatment.

Discussion

Dr. Sherman L. Shapiro: Dr. Mackay covered the uselessness of the term "dizziness" in such an excellent fashion that there is not much to say on this point. However, it is notable, that in spite of this fact, one finds increasing use of the term in titles of papers by otologists. I think this is a recognition of the fact that our classical definition of vertigo as a rotatory dizziness or even a sensation with lateropulsion tactile error is not sufficient, because there are many confused sensations which the patient describes that do not conform to either definition of vertigo. If we put the Frenzel glasses on these patients we can often display a postural nystagmus. The first thing we must do is to recognize whether the sensation is a vestibular affair by use of the Frenzel glasses and the minimal vestibular tests, but I think we must bear in mind that the essential thing in the vestibular origin is motion, either movement of objects about him or sensations produced by the patient changing his position. Here we are on pretty safe ground.

The other thing I want to say is that I heartily agree with everything Dr. Mackay said about the traumatic cases. In the patients I see, and I see a great number of them, there is just one thing I do believe that while many of these cases need the cooperation of the neuropsychiatrist, and we might be able to find an organic disturbance, or whatever has caused the neurotic or psychiatric element if there is one, the important thing is the vestibular examination using the Frenzel glasses. Using these in the examination, I believe is absolutely essential to a diagnosis.

Dr. M. M. Hipskind: Dr. Alfred Lewy wrote a long time ago, a definition of vertigo is almost as difficult to formulate as one of insanity. I believe the problem has been demonstrated tonight, that we do not have a clear concept as to the definition of vertigo. One of the definitions states that vertigo is a common and often disturbing symptom in which the senses are deceived into seeing or feeling movement. I do not think it is necessary to postulate any particular type of movement as long as there is a hallucination of movement, that is, movement that is not there. When one sees a patient with dizziness, as has already been stated, it is necessary for the clinician to have a clear understanding of what the patient means by dizziness. One patient may mean that he has a fainting sensation; another may be describing a post-in-a-hole sensation, and still another may mean true whirling, while another may mean a swimming, giddy sensation. I have found it helpful to ask the patient to tell me exactly what his sensation was and put it down in the history, using his particular expression and description of the dizziness.

Dr. Lindsay has pointed out a very important clinical fact, and Dr. Mackay referred to it, and one of the discussants referred to the problem of the post concussion state. I feel that one of the most important areas in establishing the diagnosis is the postural tests. As Dr. Lindsay says, in the case of the person who fell on the ice, and all of us have seen cases in which dizziness was the outstanding complaint following fracture and in the postconcussion state. Emphasis must be placed upon the postural tests. If the patient fails to volunteer the information that his dizziness is positional in nature, it will be wise for the physician to make specific inquiry relative to the presence or absence of the positional influence upon the precipitation of his attacks of vertigo. I think it is valid to say that the patient who has a labyrinthine type of vertigo has objective evidence of his subjective complaint. If he has a rhythmic nystagmus marked by a slow and quick component that is produced and reproduced time and again upon assuming a particular position, you have objective evidence of the patient's subjective complaint.

I do not believe that any of us would have difficulty with the problem of labyrinthine hydrops, particularly as outlined by Dr. Lindsay, because this is a recurrent type of dizziness which is usually ushered in by a sense of fulness in the ears, tinnitus, and dizziness which is episodic in nature, and the audiograms show a progressive hearing loss, starting with the low tones and involving all tones and increasing toward the high end of the scale as in nerve deafness.

Dr. Mackay has said that these attacks of dizziness stopped after a certain time, but it has been my experience that these patients go on with a persistence of the dizziness, and a loss of hearing which is irreversible. He mentioned labyrinthine hydrops and said that adequate diet will reduce the attacks of dizziness. Dr. Lindsay did not say that it sometimes becomes necessary in the treatment of the patient complaining of dizziness when all medical measures have failed, to interfere with that disturbed labyrinth. There is no condition in the treatment of injury or disease that represents such a miserable situation for the patient who has the insecurity produced by recurring attacks of dizziness. He is incapacitated and does not trust himself out of his chair. He has turbulent eye movements which are enhanced by the slightest change in position. This represents one of the really serious challenges to medicine. When these patients fail to respond to a medical regimen, some form of surgical intervention within the labyrinth—either via a transtympanal approach through the stapes and ablation of the membranous labyrinth or through labyrinthotomy destruction of the labyrinth—becomes necessary. This, of course, destroys hearing but gives stability to the patient, freeing him from his vertiginous attacks, thereby permitting him to return to normal activity.

Fortunately Ménière's disease is usually unilateral. However, the surgeon must anticipate that in about 15% of cases it may be bilateral.

Dr. Mackay has made us conscious of the dangers of measles, and I would add mumps, which is also destructive to the labyrinth. Fortunately, Dr. Mackay has given us a complete understanding of the problem in which dizziness is one of the main symptoms, infratentorial lesions, and I would emphasize and ask you to remember his remarks about the positional nature of those particular lesions. He has also described the vascular lesions, and the various diseases which may cause dizziness, although rarely, such as disseminated lupus, multiple sclerosis, and epilepsy.

Finally, I think that the clinician needs to have a working approach to this very little understood problem. In 1948 Hallpike and Dix reported that they had seen a number of cases which were septic and had a positional vertigo which they referred to as a vestibular neuronitis. The vestibular neuronitis cases had an elevated white blood cell count and sedimentation rate, and attacks of dizziness always followed some septic process. It is interesting that the dizziness subsided when the septic focus was identified and eradicated. A second type of dizziness which the classification includes has been aptly described as paroxysmal positional vertigo. In this group of cases a simple postural test will point to the diagnosis. The patient is placed upon a table and from a sitting position is taken to the horizontal plane with the head turned to one side and slightly below the level of the shoulders. Should this position produce nystagmus, the duration and type are noted. It will be found that the nystagmus is violent but rapidly subsides. When the patient is returned to the perpendicular position and again placed in the same position that produced the nystagmus, it will be found that the nystagmus does not recur. This is classified as readily fatigable and is diagnostic of the chronic paroxysmal positional type of dizziness. In these cases the dizziness could not be reproduced until the patient had a period of at least 30 minutes' rest. This has been regarded as a lesion of the otoliths.

DR. NICHOLAS TOROK: I noted with interest that Dr. Mackay introduced his very interesting presentation by saying that the word "dizziness" has actually no meaning whatsoever. It was apparent also that Dr. Lindsay felt it better to abandon the topic, and in his presentation spent the entire time just bringing out certain interesting pathologic vestibular conditions. If you follow the titles of presentations, discussions, panel discussions, and even postgraduate courses, you will notice how often the "problem of the dizzy patient" and the "management of the dizzy patient" is mentioned.

I feel we should abandon this terminology because it really only increases the confusion which exists about it, and that is not our objective. We would rather have discussion of those lesions of the vestibular apparatus where dizziness is a major or dominant symptom. It can facilitate the evaluation if the vestibular or central nervous system pathology is available.

Dr. Mackay mentioned the possible prevention of seasickness by the use of the antihistaminic drugs. I have certain doubts that they would be of great help. I would like to refer to an article published from the Netherlands a few years ago about seasickness. The whole article is devoted to a survey of members of the Dutch Navy, thousands of persons. The authors made a study, and they divided the subjects into three groups as follows: One third of all those examined didn't get any medicine; the second group received a placebo, and the third group got the various so-called antidizziness drugs, such as Bonamine, Merazine, Dramamine, and so forth. What the investigators found was interesting and was roughly as follows: In the first group, which was not given medicine at all, there was about the same percentage of persons with real seasickness. However, it was a little larger percentage than that from the other groups, who got drugs or placebos. There was no appreciable difference between the group which got the placebos and the group who received Dramamine and the other drugs. This is a very definite answer as to the tremendous psychological or emotional factors which really are responsible in causing seasickness.

Dr. Roland P. Mackay: May I ask Dr. Lindsay two questions? Why is it that in at least two-thirds of the cases of postconcussion vertigo we can never find nystagmus?

In the second place, keeping in mind the almost interminable duration of the postconcussion syndromes, what is the nature of the pathological change responsible in such cases?

Dr. John R. Lindsay: The first question was about examining for nystagmus. I use glasses with +20 d. lenses, either Bartel's glasses or goggles with +20 d. lenses. Inhibition of the nystagmus by fixation is thereby prevented. I think they are invaluable when you are looking for nystagmus, but as a general rule in a patient who has real postural vertigo with positional nystagmus, you seldom need glasses to see it.

If you have a patient with postconcussion syndrome in a fairly mild stage, you may have trouble reproducing the nystagmus and the test should be repeated if necessary. When the patient tells me that he is dizzy when he gets into certain positions and can describe those positions or movements accurately, it is a reliable indication of a vestibular disturbance. However, as the disturbance becomes milder, there may be difficulty in reproducing the nystagmus on postural tests. There is no question that there are patients who continue to have symptoms for other reasons, but a careful history will usually indicate those who have true postural vertigo. The use of Bartel's or Frenzel's glasses is a necessity in my experience.

Dr. Shapiro mentioned the necessity of reproducing the nystagmus in order to have the objective evidence that an organic basis exists for the complaint.

Abstracts from Current Literature

Ear

ADVANCES IN THE STAPES MOBILIZATION FOR OTOSCLEROTIC DEAFNESS. ALAN AUSTIN SCHEER, New York J. Med. 58:191 (Jan. 15) 1958.

Scheer commends the operating microscope which now permits safe and accurate work, and has contributed to a marked increase in the number of successful results. It helps to observe the footplate in all details, especially the posterior crus and adnexa, also to observe the anterior crus and attachment to the footplate. The elevated otosclerotic incus is visualized, and the incudostapedial joint is in full vision as well as the round window membrane. The facial nerve route and the capillary vessels are seen. It is no longer necessary to use the binocular loop. As soon as the local anesthetic is effective, the microscope is swung into place. The 6-power enlargement is used for the incision and exposure of the middle ear, the 10-power is adjusted, then the 16 or 25 magnification is set according to need. The latter is seldom needed. Any higher power narrows the field too much. The details of mobilization cannot be described here but should be studied from the author's original. Suffice it to say that "the majority of cases do not have the presence of a true otosclerotic mass." Blood which accumulates on the footplate must be removed since the surface of the footplate must be seen in detail during efforts to free the stapes. Suction is paramount, and no complications.

Formerly some 33% of Scheer's patients had improved hearing; now this has been increased to 80%! "Stapes mobilization has become the permanent primary treatment for otosclerotic deafness."

I. W. VOORHEES, New York.

THE PRINCIPLES AND PRACTICE OF BONE CONDUCTION AUDIOMETRY: A REVIEW OF THE PRESENT POSITION. J. D. HOOD, Proc. Roy. Soc. Med. 50:689 (Sec. of Otol. pp. 13-21) (Sept.) 1957.

Whereas the technique of air-conduction audiometry is well established, the basic principles of bone-conduction audiometry are still far from being generally understood. In consequence, the tests are still too often inefficiently performed and hence fail to provide the vital diagnostic information which is their purpose. This disappointing state of affairs is unnecessary and requires for its correction no more than a rational standardization of technical equipment and test procedures, whereby proper use is made of the considerable body of acoustic and physiological knowledge now at our disposal. The principles underlying the need for masking in bone-conduction audiometry are first discussed, and the basic requirements of the masking noise described. Fletcher's critical band width concept is used to determine the physical characteristics of narrow band noises which confer upon them maximum efficiency for the masking of pure tones of specified frequencies. An important feature of these bands is that their masking effect is equal to their sensation level in decibels. This enables step by step masking to be carried out in what is termed the "shadowing procedure," so that the true bone conduction threshold is accurately determined by the "change over" point at which the bone-conducted tone can be conclusively demonstrated to be heard in the tested ear.

The masking is applied by means of an insert receiver, thus increasing the interaural attenuation of the head, and hence extending the limits of the tests.

The technique described brings within the resolving power of air- and bone-conduction audiometry all but a few exceptional cases.

HOOD, London.

RECENT WORK ON THE BIOCHEMISTRY OF THE LABYRINTHINE FLUIDS. L. CITRON and D. EXLEY, Proc. Roy. Soc. Med. 50:697 (Sec. of Otol. pp. 21-25) (Sept.) 1957.

Study of the chemical composition of the labyrinthine fluids is considered to be the essential first step in seeking a satisfactory explanation of the known circulatory derangements of conditions such as Ménière's disease and the Scheibe type of hereditary cochleosaccular degeneration. Observations of earlier workers are recorded—Shambaugh, Guild, and Secretan—and their results indicated. Microchemical analysis of mammalian labyrinthine fluids was first carried out by Smith, Lowry, and Wu (1954), and the present investigations repeat and extend their observations.

The methods of collecting specimens of perilymph and endolymph from the cat and the guinea-pig are indicated, with photomicrographs and new methods of microchemical analysis detailed.

Tables of results are given, comparing the concentrations of various ions in serum, cerebrospinal fluid, perilymph, and utricular endolymph and cochlear endolymph, and particular attention is drawn to the peculiar composition of endolymph. It is suggested that this is dependent upon an "active transport" mechanism, which becomes deranged in Ménière's disease, with resultant disturbance of the endolymph circulation.

CITRON, London.

CANCER OF THE EAR. P. GUNS and J. C. MINET, Ann. oto-laryng. 74:904 (Dec.) 1957.

The authors present 128 cases of cancer of the ear that were observed during the last 13 years: 105 involved the auricle; 5 were retroauricular; 9 involved the external auditory canal, and 9 the middle ear.

Of those involving the auricle, 74 were spinocellular, 14 simple basocellular, 1 pigmented basocellular, and 1 papillary basocellular. In the retroauricular region there were one spinocellular and two basocellular epitheliomas. In the external canal there were four spinocellular, one from a sebaceous cyst, and two basocellular epitheliomas. In the middle ear there were eight spinocellular and one case of basocellular epithelioma.

Nine cases involving the middle ear and three cases of the external canal are presented in detail. The survival mean is from 15 months to two years. The therapy is both surgery and radiation. However, many of these growths are radioresistant. Early diagnosis is stressed.

PERSKY, Philadelphia.

ACUTE SUPPURATIVE OTITIS MEDIA IN ADULTS. J. P. MEUNIER and J. L. MEUNIER, Ann. oto-laryng. 75:16 (Jan.-Feb.) 1958.

The authors review 1000 cases of acute suppurative otitis media that were hospitalized from 1946 to 1956. There were only 15 patients that required mastoidectomy. They had two cases of labyrinthitis, two cases of facial paralysis, and three cases of meningitis.

They conclude that acute suppurations in the adult is a very benign infection and can be cured by conservative measures, if the patient is hospitalized, under daily supervision, and the proper and adequate drainage of the middle ear is established and maintained. Attention to the rhinopharynx is very important. Antibiotic therapy is of little significance, since it was given in only 29% of their cases, and only because of a suspected complication.

Mastoiditis developed in 2.2% of the cases, and there were no fatalities.

PERSKY, Philadelphia.

Larynx

FLEXIBLE ESOPHAGOSCOPY. EMANUEL FRIEDMAN, DAVID KATZ, and SIDNEY SELESNICK, New York J. Med. 58:351 (Feb.) 1958.

From the Medical Service of the Veterans' Administration Hospital, West Haven, Conn. and the Yale University School of Medicine, the authors recite their experiences with the flexible esophagoscope in 225 procedures. A plea is made for direct tube work by the gastroenterologist; in fact it is stated that the movement has been gathering momentum since 1947. Fear of perforation of the esophagus at the level of the cricopharyngeus has restrained many would-be enthusiasts; therefore the invention of the soft-nose instrument which has been favored by Schindler, Boros, and Hufford, working separately or together. Preliminary medication with phenobarbital, morphine, and atropine was used in all cases. The patient was placed in the left lateral position, and anesthesia was obtained by swallowing Tetracaine as recommended by Selesnick. There were 94 "negative examinations" done for such symptoms as "heartburn" and "dysphagia." The Schindler, Boros, and Hufford instruments were tried, and there was a preference for the Hufford, which "is outstanding for its facility of passage." Moreover, it has an obturator, the length of which can be adjusted to suit, and there is space for suction and a distal light. It can be used in the OPD service or at the patient's bedside. Since asepsis is not paramount, the O. R. need not be demanded. So far there has been no morbidity and no mortality. "It is simple and safe." Eight references are cited, and a table shows the results on 153 patients.

I. W. VOORHEES, New York.

OBSERVATIONS UPON THE EXPERIMENTAL REPAIR OF THE TRACHEA USING AUTOGENOUS AORTA AND POLYETHYLENE TUBES. J. J. PRESSMAN and M. B. SIMON, Surg. Gynec. & Obst. 106:56 (Jan.) 1958.

Resections of the complete circumference of the cervical trachea of such length as to make end-to-end suture impossible require an indwelling prosthesis to bridge the defect. The most satisfactory material from which to make such a prosthesis seems to be polyethylene (or tygon) tubing. Because of the distribution of blood vessels to the tracheal rings, this prosthesis must be telescoped into the lumen of the tracheal segments rather than vice versa. When so inserted, the tubing has a tendency to be displaced downward and to occlude respiration. This mishap may occur at any time for many months postoperatively. In large measure, however, it can be avoided by using stainless steel wire fixation sutures, No. 24 gauge or heavier, placed through the muscles of the neck, the residual tracheal wall, and the polyethylene tube, and drawn out of the muscles on the opposite side. The sutures must then be tied or twisted across the midline and buried or fastened to buttons externally. The use of such a prosthesis requires careful planning to avoid too long a tube, the result of which may be the formation of occluding granulomas at the laryngeal level or interference with swallowing at the thoracic inlet.

The occurrence of granulations at either end of the prosthesis is stimulated by any untoward degree of movement of the tube. It is lessened by proper fixation of the tube and the use of an aortic homograft as a patch closing the defect. A fibrous pseudotrachea forms, which ultimately epithelializes throughout its luminal surface. It undergoes contraction in a circumferential and longitudinal direction of such magnitude as appreciably to stretch the remaining segments of the trachea above and below the defect. The tube thus formed never gains a sufficient degree of rigidity to remain patent during inspiration. This latter shortcoming necessitates the continued presence of an indwelling polyethylene prosthesis, but there is evidence at hand to indicate that contraction of the fibrous tube that forms about the polyethylene may be of sufficient magnitude to permit, in some cases and after a period of years, an end-to-end anastomosis of the upper and lower tracheal segments, whereas this was not possible initially. This result is produced by the stretching of the membranous rings. There is some disagreement as to whether or not the polytheylene tube, because of the absence of normal cilia, hinders the escape of pulmonary and bronchial secretions, but it is generally agreed that polyethylene is less objectionable in this respect than metallic tube inserts.

AUTHORS' SUMMARY.

TUMORS OF THE LARYNX AND LARYNGOCELES. J. LEROUX-ROBERT and A. ROGEON, Ann. oto-laryng. 74:885 (Dec.) 1957.

The authors discuss the problem of laryngocele, which is a dilatation of the appendix of the ventricle of Morgagni. There are two types—the internal, which extends into the larynx, ballooning out the ventricle, the aryepiglottis folds, and sometimes the vallecula, and the external type, which extends laterally in the neck through the thyrohyoid membrane.

Six cases are presented that are associated with a carcinoma of the larynx. It is the opinion of the authors that the malignancy is the cause of the laryngocele in this group of cases. The diagnosis of these hernias of the larynx is often difficult. Frontal tomography is essential to help establish this with certainty.

PERSKY, Philadelphia.

Nose

CORRECTIVE SURGERY OF THE NASAL SEPTUM. AUBRY and J. GAREZ, Ann. oto-laryng. 75:5 (Jan.-Feb.) 1958.

Septal perforations are usually postoperative. While occasionally they are the results of faulty technique, more often they are due to marked septal ridges, where it is most difficult to elevate the mucous membrane without producing a perforation. If the tear in the mucous membrane on one side can be opposed to an intact area of the opposing membrane, a perforation can be avoided. Perforations may also occur secondary to infection of the septum due to the packing.

In dealing with small perforations the authors freshen the edges of the perforation. An incision is made 1 cm. posterior, and the mucous membrane is freed and separated for some distance posterior. Then either a sliding or a VY-flap is brought forward to cover the perforation. One or two small sutures are inserted into the anterior edge of the scarified perforation.

Larger perforations are closed either by taking a flap from the inferior turbinate and so producing a synechia which later is severed, or by making a typical Indian flap from the inferior turbinate. A large rectangular flap is cut from the inferior turbinate, with its posterior end remaining attached. This covers the perforation. Two to three weeks later the posterior end is severed.

In larger perforations and where there is a deformity of the nasal pyramid, a rhinoplasty is performed, at which time the edges of the perforation can be approximated, depending on its size and position.

PERSKY, Philadelphia.

Miscellaneous

ANTIMICROBIAL TREATMENT OF UPPER RESPIRATORY INFLAMMATION. CARL A. BERNTSEN JR., New York J. Med. 58:195 (Jan. 15) 1958.

Bernsten works in the Department of Medicine at Cornell, and in Bellevue Hospital. He thinks that "antimicrobial drugs are useful in only a small fraction of respiratory inflammations." From 1951 to 1953, the Commission on Acute Respiratory Diseases studied some 3000 persons divided into groups (*J. A. M. A.* 133:588, 1947). Some 75% to 80% had a "brief and benign illness" which seemed not to be greatly influenced by drugs. The ineffectiveness of antimicrobial drugs was stressed. But clinical rather than laboratory criteria must be our guide. Penicillin in streptococcic infections often gives dramatic results regardless of aspirin or quinine or other old stand-bys. Both streptococci and pneumococci are common invaders which quickly succumb to penicillin. Three intramuscular injections of 300,000 units at three-day intervals are effective against streptococci, but to minimize occurrence or relapse the administration had better continue for 10 days or more, according to the case under care. Chloramphenicol is often useful where other antibiotics seem to have failed, but it should not be given without an eye on its selective action on bone-marrow deterioration. One may be obliged to work "trial and error" in resistant infections. Experience must guide the physician in all such instances. Masking of symptoms may be dangerous because of delayed dangerous pathologic processes going on surreptitiously, especially in bone (mastoiditis).

ABSTRACTOR'S COMMENT: It is surprising how the word "inflammation" has persisted in the literature of medicine from the *inflammatio* of the Latin writers in early days, centuries before Pasteur and the germ origin of many diseases was established. *Inflammare* means to set on fire. Any reaction to irritation or trauma sets up swelling, redness, heat, hyperemia, stasis, and changes in the walls of blood vessels which permits exudation of serum, etc. Many excellent writers in modern medicine use the word inflammation when they mean infection by microorganisms. One may, to be sure, have inflammation without infection, but this must be rare. If it occurs as a result of some bodily insult, it is commonly followed by bacterial activity, which often calls for surgery, or, in these days, antimicrobial agents. In the interest of accuracy we, all of us, need to be more guarded in the use of "inflammation."

I. W. VOORHEES, New York.

THE VALUE OF SURGERY IN THE TREATMENT OF NASAL ALLERGY. RUSSELL CLARK GROVE, New York J. Med. 58:359 (Feb. 1) 1958.

Grove works on the ENT Service and in the Institute of Allergy in Roosevelt Hospital, New York City. He believes that we should take a very careful history as to allergy in all patients with hypertrophy or hyperplasia of the mucous membrane. In fact every means known to medicine should be tried out before resorting to surgery because results may not be as good as hoped for, thus disappointing the patient and surgeon alike. Complete laboratory diagnosis calls for urine, blood, and possibly basal metabolism studies. As everyone knows, etiology may be annoying in its persistent elusiveness. "We do not favor either electrical or chemical cauterization." Crusting and atrophy may follow and be a permanent annoyance. . . . Chronic hyperplastic sinusitis may exist with or without polyps. Suppuration is not commonly present. Polypi were seen in a little over one-half of the patients, and was usually bilateral (unilateral in 11). . . . Treatmem for allergy by vaccines, antibiotics and corticosteroids is recommended with or without surgery, and must be carried out for some time. The patient must be the judge as to relief obtained. The word "cure" must be used guardedly. Recurrences are to be expected in many cases. Therefore patience and fortitude are demanded from patient and doctor. There are three references to the literature, but no "pictures."

I. W. VOORHEES, New York.

ADVANCES IN THE DIAGNOSIS AND TREATMENT OF ACUTE PYOGENIC MENINGITIS. EMANUEL APPELBAUM and CHARLES ADLER, New York J. Med. 58:363 (Feb. 1) 1958.

This is Part II under the same heading as Part I previously abstracted in the ARCHIVES. The report comes from the Bureau of Laboratories, New York City Department of Health. It is an extension of the study of pneumococcic meningitis, that type due to Hemophilus influenzae, to streptococci, staphylococci, and unusual forms, or miscellaneous. The fulminating form is characterized by overwhelming bacteremia and profound circulatory collapse; therefore drugs should be promptly given to prevent the latter, while the laboratory is seeking the causative germ (not always the Meningococcus). Chemotherapy and the antibiotics save many patients now who would have died in other days. When Pneumococcus is present, penicillin is the drug of choice, administered by needle. The H. influenzae is sensitive to streptomycin or chloramphenicol or the tetracyclines. Therefore one has to know the enemy before choosing the proper ammunition against him. The sulfonamides, of course, may prove very useful, depending on the causative germ. Seven tables and 80 references elucidate this very careful and complete study of a dangerous and death-dealing infection.

ABSTRACTER'S COMMENT: There may be so little time after the patient is first seen. Removal to a hospital at once is imperative. Pending that, one must inject penicillin or whatever agent is at hand. Precious hours may be lost while waiting for laboratory reports. Blood and spinal fluid are, of course, the first to be studied. The portals of entry, nose and throat, are under suspicion. They should be "sterilized" insofar as possible.

I. W. VOORHEES, New York.

TUMORS IN CHILDHOOD. HAROLD W. DARGEON, New York J. Med. 58:402 (Feb. 1) 1958.

The author calls attention to the importance of recognizing cancer in children. "The latest mortality statistics (1947) show that next to accidents, neoplastic diseases, which include leukemia and benign and malignant tumors stand first among all causes of death in the group from 5 to 14 years of age." Lymphosarcoma, for example, may originate in the nasopharynx, or cervical glands, and may not be quite manifest until it makes pressure on the airways. The symptoms of rheumatic fever may becloud an actual malignancy. Besides blood studies, one now tests bone marrow (from the sternum). Surprises are not uncommon. The author takes no fatalistic view: "Treatment either by surgery and/or irradiation continues to show gratifying results in more and more cases. . . . Earlier diagnosis of juvenile cancer can be made, and the salvage of these children will increase significantly when this is accomplished."

ABSTRACTOR'S COMMENT: ENT men know as well as anyone that cancer strikes any and all ages, and yet we may not think of it when a baby is brought in with a swollen face, or glands in the neck, or "enormous tonsils." I have seen "peritonsillar abscess" incised several times, but "no pus was found." The child had sarcoma in the left tonsil! Beware of excessive unilateral enlargement, and have all such specimens sectioned.

I. W. VOORHEES, New York.

RECENT ADVANCES IN MEDICINE AND SURGERY: SURGERY OF THE THYROID GLAND. JOSEPH N. ATTIE, New York J. Med. 58:703 (March 1) 1958.

This is Part II of a study of the thyroid with the general title of Recent Advances in Medicine and Surgery, under the editorial supervision of Robert Turell, M.D. Attie reviews in general the various nonsurgical methods of dealing with conditions of the thyroid, but with special consideration of carcinoma. He says: "It is generally conceded that the only curative treatment for thyroid cancer is total surgical ablation." And by ablation he means complete removal." . . . Recurrences are frequent when subtotal thyroidectomies are performed," for which statement he quotes Crile Jr. in support. However, he says, "Attempts at surgical extirpation should be made only if the lesions are of limited extent." As for radioiodine treatment, it should be advocated only in patients below 40 years, and never during pregnancy or lactation.

Hyperthyroid patients may be grouped as mild and severe in re preoperative management. The mild cases may be prepared by administering Lugol's solution, or saturated K9 for two or three weeks. Severe conditions may require propylthiouracil in doses from 300 to 1000 mg. daily until the active symptoms of hyperthyroidism are overcome on an OPD basis. Admission to hospital should be granted one or two days prior to surgery. Technique calls for a transverse incision. The amount to be removed is still *sub judice*, since no rule is available.

The smaller the residual left in situ, the less likely is recurrence. It has been estimated at 0.8 to 1.0 gm. One should dissect the recurrent laryngeal free and keep it under observation until the dressing is to be applied.

To summarize: Hyperthyroidism may be managed in three ways. By antithyroid drugs, radioactive iodine, and subtotal thyroidectomy. Because of the high incidence of recurrence even after prolonged use of antithyroid drugs, they have limited use in controlling thyrotoxicosis. In a patient over 40, radioactive iodine has a definite indication in diffuse toxic goiter, in some nodular types, or in poor surgical risk cases, or in those who, outright, refuse surgery. As already mentioned, this powerful agent is not to be used in pregnant women or in the lactation period. "At the present time, subtotal thyroidectomy is indicated in toxic nodular thyroids, and in diffuse toxic goiter in patients over 40."

The author writes from experience gained in the Department of Surgery, Maimonides and Long Island Jewish Hospitals. He sets down the classifications of Lahey, Hare, and Warren (1940) which concern three groups of malignancy—I, low or potential malignancy; II, moderate malignancy; III, high malignancy. There are subgroups in each division. Also he recognizes the classification of Watson and Pool (1940), which has six subgroups. There is also that of Martin (1954) in use at Memorial Hospital, New York, and, finally, a fourth group, that of Warren and Meissner (1941), which is described as an "excellent classification," a modification of Hare and Salaman, A, differentiated carcinoma and B, undifferentiated carcinoma. Attie cites 100 references to the literature.

ABSTRACTOR'S COMMENT: There is criticism of the ENT man by general surgeons for "invading" a province other than the strictly limited specialization. However, the well-trained modern ENT man is likely to be proficient in conditions of the head and neck which will contain some brain and neurosurgery, and dissections of the collis for whatever surgical condition. The "doing" of septums, sinuses, and A. and T. only has long since been relegated to the earlier days of OALR. Progress means advancement into adjacent fields, but only after exhaustive preparation.

I. W. VOORHEES, New York.

BACTERIOLOGIC AND CLINICAL EXPERIENCES AND THE METHODS OF CONTROL OF HOSPITAL INFECTIONS DUE TO ANTIBIOTIC RESISTANT STAPHYLOCOCCI. H. T. CASWELL; K. M. SCHRECK; W. E. BURNETT, et al., Surg. Gynec. & Obst. 106:1 (Jan.) 1958.

At the present time, the most significant problem with antibiotic resistant organisms is that of the Micrococcus pyogenes var. aureus (Staphylococcus aureus). Infections with these organisms do not constitute a serious problem in the general population but do present a serious problem in hospitalized patients.

Among the problems discussed are the bacteriophage typing of the organism, nasal carriers in hospital personnel, cutaneous infections with resistant staphylococci in hospital personnel, and staphylococcic respiratory infections. Modes of transmission, methods of control, modification of hospital and operating room techniques for the control of infections are thoroughly described, and the resulting drop in infections is adequate evidence of the effectiveness of the measures employed.

Whereas novobiocin and chloramphenicol were the only two systemically administered antibiotics to which the resistant staphylococci were susceptible, the authors point out that resistance to these drugs is developing. Control of hospital staphylococci cannot be attained by the discovery of additional antibiotics but must be predicated on a return to strict sterile techniques combined with intelligent limitation of the use of antibiotic agents.

FRIEDBERG, Chicago.

News and Comment

ANNOUNCEMENTS

Postgraduate Courses at Temple University.—The following courses will be given in the Department of Laryngology and Broncho-Esophagology, Temple University Medical Center, under the direction of Drs. Chevalier L. Jackson and Charles M. Morris.

Postgraduate Course in Bronoesophagology, Feb. 16-27, 1959

Postgraduate Course in Laryngology and Laryngeal Surgery, April 13-24, 1959

The tuition fee for each course is $250.00. Application blanks and further information concerning dates of subsequent courses may be obtained by writing to Jackson-Research, Lab. 604, Temple University Medical School, 3400 Broad St., Philadelphia 40.

Course in Reconstructive Nasal Surgery.—A course in "Reconstructive Surgery of the Septum and External Nasal Pyramid," sponsored jointly by the Departments of Otolaryngology of the College of Medical Evangelists School of Medicine and the University of Southern California School of Medicine, will be given at the White Memorial Hospital, Los Angeles. The course will begin at 7 p. m. on Jan. 6, 1959, and end at 12 noon on Friday, Jan. 16. Dr. Maurice Cottle will be the director.

All applications should be forwarded to Dr. Leland R. House, Chairman, Department of Otolaryngology, The College of Medical Evangelists, 1720 Brooklyn Ave., Los Angeles 33.

Books

BOOKS RECEIVED

* **Le Cancer de la corde vocale.** By J. Piquet. Price, 1600 fr. Pp. 204, with 53 illustrations. (Monographies Oto-Rhino-Laryngologiques Internationales. Founded by M. Vernet, Published by G. Portmann, M. Sourdille, J. Terracol. & M. Vernet). Masson & Cie., 120, boulevard Saint-Germain, Paris-6e, 1958.

* **Les Sinusites de l'enfance.** By J. Terracol. and Y. Guerrier. Price, 1400 fr. Pp. 166, with 28 illustrations. (Monographies Oto-Rhino-Laryngologiques Internationales. Founded by M. Vernet, Published by G. Portmann, M. Sourdille, J. Terracol & M. Vernet). Masson & Cie., 120, boulevard Saint-Germain, Paris-6e, 1958.

Ciba Foundation Symposium: The Cerebrospinal Fluid, Production, Circulation and Absorption. Edited by G. E. W. Wolstenholme, O.B.E., M.A., M.B., B.Ch., and Cecilia M. O'Connor, B.Sc. Price, $9.00. Pp. 335, with 141 illustrations. Medical Book Dept., Little, Brown & Company, 34 Beacon St., Boston 6. Published in England by J. & A. Churchill, Ltd., 104 Gloucester Place, Portman Sq., London, W. 1, 1958.

* **Histopathologie der Tumoren der Kiefer und der Mundhöhle.** By Prof. Dr. Med. Erich Langer. Price, $11.80. Pp. 167, with 212 illustrations. Georg Thieme Verlag, Herdweg 63, (14a), Stuttgart, 1958.

* To be reviewed.

CORRECTION

Through error, in the August issue of THE ARCHIVES the American Laryngological Association was referred to as the American Otolaryngological Association on the cover, in the Table of Contents, and in Society Transactions.

Directory of Otolaryngological Societies *

FOREIGN AND INTERNATIONAL

Canadian Otolaryngological Society
(Société Canadienne d'Otolaryngologie)

President: Dr. Robert T. Hayes, 42 Cobourg St., St. John, N. B.
Secretary: Dr. Donald M. MacRae, 34 Spring Garden Rd., Halifax, N. S.

Venezuela Otorhinolaryngology Society

President: Dr. Alfredo Celis Pérez.
Secretary: Dr. Jesús Miralles.

International Association of Allergology

President: Dr. Samuel M. Feinberg, 303 E. Chicago Ave., Chicago 11.
Secretary-General: Dr. José Quintero Fossas, Paseo 313, Vedado, Havana, Cuba.
Meeting: Third International Congress of Allergology.

International Bronchoesophagological Society

President: Dr. Jo Ono, Tokyo.
Secretary: Dr. Chevalier L. Jackson, 3401 N. Broad St., Philadelphia 40.
Meeting: Seventh International Congress of Bronchoesophagology.

International College of Surgeons, Section of Ophthalmology and Otolaryngology

President: Dr. E. G. Gill, 711 S. Jefferson St., Roanoke, Va.
Secretary-Treasurer: Dr. Louis Savitt, 3403 W. Lawrence Ave., Chicago 25.

International Congress of Audiology

General Secretary: Dr. H. C. Huizing, 182 Oostersingel, Groningen, Netherlands.
Place: Leyden, Netherlands.

International Course in Paedo-Audiology

General Secretary: Dr. H. C. Huizing, 182 Oostersingel, Groningen, Netherlands.
Place: Groningen, Netherlands.

Pan-American Association of Oto-Rhino-Laryngology and Broncho-Esophagology

President: Dr. José Gros, Havana, Cuba.
Secretary: Dr. Chevalier L. Jackson, 3401 N. Broad St., Philadelphia 40.
President of Congress: Prof. R. David de Sanson.
Secretary of Congress: Dr. Walter Benevides, Caixa Postal 2838, Rio de Janeiro.

NATIONAL

American Medical Association, Scientific Assembly, Section on Laryngology, Otology and Rhinology

Chairman: Dr. Victor R. Alfaro, Washington, D. C.
Secretary: Dr. Walter E. Heck, San Francisco.
Meeting: Atlantic City, June 8-12, 1959.

American Academy of Ophthalmology and Otolaryngology

President: Dr. LeRoy A. Schall, 243 Charles St., Boston.
Executive Secretary-Treasurer: Dr. William L. Benedict, 100-1st Ave. Bldg., Rochester, Minn.

* Secretaries of societies are requested to furnish the information necessary to keep this list up to date.

American Board of Otolaryngology

President: Dr. Gordon D. Hoople, 1100 E. Genesee St., Syracuse 10, N. Y.
Secretary-Treasurer: Dr. Dean M. Lierle, University Hospitals, Iowa City.

American Broncho-Esophagological Association

President: Dr. Francis W. Davison, Geisinger Memorial Hospital, Danville, Pa.
Secretary: Dr. F. Johnson Putney, 1712 Locust St., Philadelphia 3.
Place: The Homestead, Hot Springs, Va. Time: March 10-11, 1959 (afternoon only).

American Laryngological Association

President: Dr. Fred W. Dixon, 2060 E. 9th St., Cleveland.
Secretary: Dr. James H. Maxwell, University Hospital, Ann Arbor, Mich.
Place: The Homestead, Hot Springs, Va. Time: March 8-9, 1959.

American Laryngological, Rhinological and Otological Society, Inc.

President: Dr. Gordon D. Hoople, 1100 E. Genesee St., Syracuse 10, N. Y.
Secretary: Dr. C. Stewart Nash, 708 Medical Arts Bldg., Rochester 7, N. Y.
Place: The Homestead, Hot Springs, Va. Time: March 10-12, 1959 (mornings only).

SECTIONS:

Eastern.—Chairman: Dr. Joseph L. Goldman, New York.
Place: The Savoy Plaza, New York. Time: Jan. 8-9, 1959.
Southern.—Chairman: Dr. James T. King, Atlanta, Ga.
Place: Hotel-Atlanta Biltmore, Atlanta, Ga. Time: Jan. 23-24, 1959.
Middle.—Chairman: Dr. Alfred J. Cone, St. Louis, Mo.
Place: Park Plaza Hotel, St. Louis. Time: Jan. 12-13, 1959.
Western.—Chairman: Dr. Robert C. McNaught, San Francisco, Calif.
Place: The Ahwahnee Hotel, Yosemite Valley, Calif. Time: Jan. 17-18, 1959.

American Rhinologic Society

President: Dr. Russell I. Williams, 408 Hynds Bldg., Cheyenne, Wyo.
Secretary: Dr. Robert M. Hansen, 1735 N. Wheeler Ave., Portland, Ore.

American Otological Society, Inc.

President: Dr. Moses H. Lurie, 483 Beacon St., Boston.
Secretary: Dr. Lawrence R. Boies, University Hospitals, Minneapolis 14.
Place: The Homestead, Hot Springs, Va. Time: March 13-14, 1959.

American Otorhinologic Society for Plastic Surgery, Inc.

President: Dr. Armand L. Caron, 36 Pleasant St., Worcester, Mass.
Secretary: Dr. Joseph G. Gilbert, 75 Barberry Lane, Roslyn Heights, N. Y.

American Society of Facial Plastic Surgery

President: Dr. Trent W. Smith, 327 E. State St., Columbus 15, Ohio.
Secretary: Dr. Samuel M. Bloom, 123 E. 83rd St., New York 28.
Meeting: 1313 Bedford Ave., Brooklyn, Nov. 12, 1958.

American Society of Ophthalmologic and Otolaryngologic Allergy

President: Dr. Joseph W. Hampsey, Grant Building, Pittsburgh 19.
Secretary-Treasurer: Dr. Daniel S. DeStio, 121 S. Highland Ave., Pittsburgh 6.

Otosclerosis Study Group

President: Dr. Lawrence R. Boies, University Hospital, Minneapolis.
Secretary-Treasurer: Dr. Arthur L. Juers, 611 Brown Building, Louisville.

Society of Military Otolaryngologists

President: Lt. Col. Stanley H. Bear (MC), USAF, Maxwell AFB, Ala.
Secretary-Treasurer: Capt. Maurice Schiff (MC), U. S. N., U. S. Naval Hospital, Oakland, Calif.

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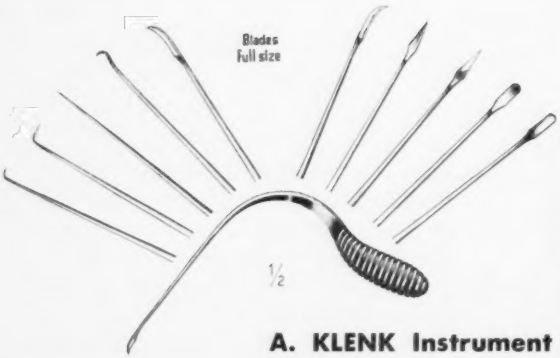

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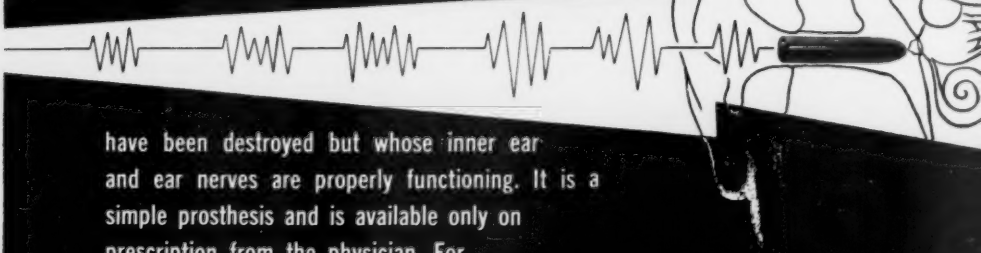

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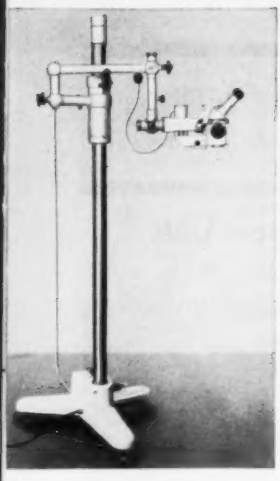

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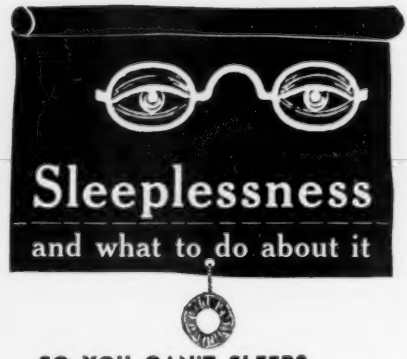

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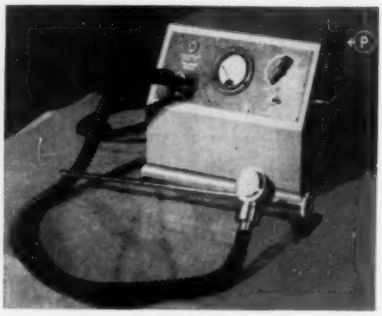

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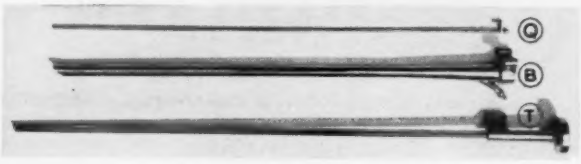
luminator (L)
power unit (P)



Optics, certified by the Optical Institute, are imported from France.

The bronchoscope is of special design with light carrier canal inside the tube. This canal accommodates the standard Jackson light carrier and lamp, as well as the new quartz rod—thus the bronchoscope may be used in a routine fashion for regular procedures, or, with a quartz rod for photography.

The quartz rod is specially treated to transmit maximum light from the luminator to the distal end of the tube. These rods are encased in BRONCHALLOY, giving them a strength which belies the delicate design.

The forward-viewing telescope is complete with an anti-fogging device: an air jet across the face of the distal telescope lens assures constant clear vision. Adaptor rings are available to make proper contact between the proximal telescope eye-piece and the camera lens.

The luminator is the heart of the instrument . . . engineered so that its use is simple but very effective. The lamp in the luminator produces excellent light transmitted distally by the quartz rod. It is air-cooled by a large capacity pump housed in the power unit. Rheostat controls on the power unit provide variable light—four volts for regular bronchoscopy, up to 10½ volts for "over-illumination" of the lamp for the white light ideal for picture taking.

References:

Jackson, C.L.: New Instrumentarium for Peroral Endoscopy and Endoscopic Photography. LARYNGOSCOPE, *67*:777, (Aug.) 1957.

Fourestier, M., Gladu, A., and Vulmiere, J.: LA PRESSE MEDICALE, *60*:1292, (Oct.) 1952.

Dubois De Montreynaud, J.M., Edwards, Roger, J., and Gladu, Amedé J.: LARYNGOSCOPE, *66*:637, (June) 1956. ——JOUR. BIOLOG. PHOTOG. ASSOC., *24*:45, (May) 1956.

The Jackson Fotobronchoscope is on display in Pilling's Philadelphia store and New York office. Write Philadelphia for further information and prices; send orders to Philadelphia.

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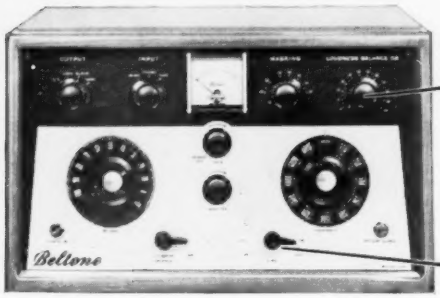
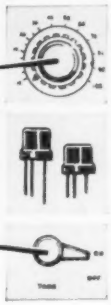


Model 14-A, $595. Weight, 26 lbs. Dimensions: 21¼" Long x 13¼" High x 13¾" Deep.



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